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No. 4.—"Q" FEVER: THE DOMESTIC ANIMAL AS A SOURCE OF INFECTION FOR MAN*

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There was again a large attendance in the Royal Hall, Harrogate, for the consideration of the paper presented to the annual Congress of the Association by Dr. Slavin, on the subject of "Q" fever, on the morning of Thursday, September 11th, 1952. This session was presided over by Mr. H. B. Allan, M.R.C.V.S., D.V.S.M., of Harrogate, with Dr. K. C. Sellers, PH.D., B.SC., M.R.C.V.S., D.V.S.M., of Headingley, Leeds, as Recording Secretary.

The CHAIRMAN: Ladies and gentlemen,—We are going to have a busy morning and I want now to introduce to you Dr. Slavin. I think many of you know him quite well and I think that is most gratifying to have at this Congress a paper on a subject about which many of us have not, shall I say, a very deep knowledge. All the same, this subject is one which keeps popping up in various periodicals and journals and in our travels we are coming across it. This is a golden opportunity for having a paper such as this in a very compact form and I think Dr. Slavin should be congratulated for presenting such a paper. I will now ask Dr. Slavin if he would care to elaborate on the paper.

Dr. G. SLAVIN: People still ask "What is 'Q' fever?" I hope this paper will have answered most of these questions. Derrick in Brisbane first called it "Q" fever and "Q" fever it has remained. Derrick and the medical practitioners who first noticed this disease deserve great credit for acute clinical observation for deciding that it was a new entity. As well as the numerous occupational diseases to which these men were subject there were numerous local fevers to confuse the picture. Large-scale outbreaks of any disease can hardly escape notice, but these were sporadic cases. Derrick's work in establishing the existence of the infective agent is also a good illustration of the way to tackle such problems. With their knowledge of rickettsial diseases the Australian workers naturally looked for a tick as vector and by one of those fortunate chances which have characterised the work on this disease, quickly found both a vector and a natural host. Later work has diminished the importance of the tick but has not altered the soundness of their original observations.

The reasons for the isolation of *R. diaporica* in U.S.A. were, of course, that other rickettsial diseases were being studied at that time. *R. diaporica* was regarded as interesting but of no particular importance. "Q" fever stayed at this stage until the latter part of the 1939-45 war when someone was astute enough to suspect *R. burneti* infection as the cause of non-specific pneumonia occurring in Allied troops in Italy although the increasing precision of diagnosis would have made its discovery inevitable.

Following the first discovery of the disease in man and cows in England, it was decided to investigate the incidence in domestic animals. These animals do not themselves suffer ill effects from the infection but must play some part in its continued existence or at least indicate its presence and, therefore, the possibility of human infection. Our animals can produce the organism in vast quantities especially in the placenta and with its great resistance to drying, combined with the habits of cows, dissemination of the organism is assured. It does seem extraordinary that the heavy growth of *R. burneti* in the placenta does not interfere in any way with the nutrition of the foetus. Infection of the udder is prolonged and we have records showing persistence for nearly three years.

There is evidence now that "Q" fever infection in the human is not quite so harmless as it once appeared to be.

Outbreaks of "Q" fever in the human are rare in this country but there must be many sporadic cases. The evidence for the origin of any particular case has sometimes to be stretched a little to cover the facts, but cases have an irritating habit of turning up in places where infection is improbable. In one American outbreak the only common factor was a barber's shop. It is too much to expect a doctor in general practice to differentiate this fever from the numerous other fevers such as colds and influenza. A ferocious headache seems to constitute the main difference but my personal experience is that severe headaches do occur after recent contact with animals, which are quite definitely not "Q" fever. What they are is another problem to unravel.

Slides

There is a great variation in the amount of infection in various counties, but as stated in the paper there are weighting factors. Some counties have been entirely missed but this is because samples were not available.

The first map shows the location of the farms from which samples have come. Some of these dots represent more than one sample. A correct interpretation of this map would require another showing the distribution of milking cattle. The second map shows the location of farms on which *R. burneti* infection has been found. The figures do not show the extent of infection on these farms but from those available the number of infected cows on a farm appears to be small. Table II in the paper shows the blood titres of some infected cows and the results of the biological test of milk samples from these cows. By an unfortunate error the printer omitted the lines, making this table almost unintelligible.

As regards the actual test a note of technique is given for record purposes. The first antigens prepared at Weybridge and a number of others were liable to give non-specific reactions. It is certain that a reaction obtained with the antigen now issued is specific and is a true indication of past or present infection.

Dr. Slavin's paper was as follows:—

In 1935 Derrick, of the Public Health Department in Brisbane, investigated cases of fever which had begun about two years earlier among workers in a large meat-works in Brisbane. Up till then only about 20 cases had occurred in about 800 workers but some medical practi-

*Presented to the 70th annual meeting of the British Veterinary Association, at Harrogate, on September 11th, 1952.

tioners were of the opinion that this was a new disease. Blood cultures and serological tests for known pathogens were negative. The possible causes investigated were typhus (several forms of which are endemic in Queensland), undulant fever (a recognised occupational disease of meat workers), aberrant typhoid and paratyphoid fevers and leptospirosis. Other animal diseases were also considered and excluded.

As there is still some misunderstanding about the origin of the name "Q" fever, Derrick's actual words may be quoted here: "Then the suspicion arose and gradually grew into a conviction that we were dealing with a type of fever which had not previously been described. It became necessary to give it a name and 'Q' fever was chosen to denote it until fuller knowledge should allow a better name." Fuller knowledge has not produced a better name and the useful term "Q" fever is now reserved for this disease.

With blood or urine from patients Derrick was able to establish an infection in guinea-pigs which caused a characteristic fever reaction. He was further able to show that following this fever reaction the guinea-pigs became immune to another injection of the same infective agent.

At this point Burnet and Freeman (1937) took over the investigation and proceeded to examine further the characters of this infective agent. It would infect not only guinea-pigs but also mice and albino rats and survive in the chorio-allantois of chick embryos. It was also filterable through a gradocol membrane with 0·7μ pores but not reliably so. The only animal to show any lesions was the mouse, in which there was a distinctive enlargement of the spleen. In mouse spleen stained with haematoxylin and eosin there were oval areas about the size of a nucleus which were filled with a lightly stained material of faint, uniformly granular texture. Special staining showed these granules to be rickettsiae. Rickettsiae in relatively pure form were obtained from mouse spleens by differential centrifugation and suspensions of them were agglutinable by sera from experimentally infected animals and from human patients.

Sources of infection were considered and it was concluded that the most likely for the abattoir worker was tick faeces on the cattle and an extensive search was made for a vector. Ectoparasites were collected from a wide range of wild and domestic animals in the area from which the infected cattle came. On six occasions, infected parasites—all of which were ticks—were found on bandicoots captured at Moreton Island. The tick—*Haemophysalis humerosa*—is mostly a parasite of bandicoots and opossums: it has also been found on two species of rat, on cattle and once on a horse. It has also been taken from the dollar bird, *Eurystomus orientalis*. The bandicoot is a small marsupial and is notorious for the number and range of its parasites. With the evidence available at that time a great deal of importance was attached to the tick as a vector. However, it is likely that this rickettsia, named *R. burneti* by Derrick, is a universal parasite with a very wide host range from ticks to man.

At about the same time, there was isolated from ticks from Montana in U.S.A. a hitherto unknown rickettsia which was remarkable chiefly because it was filterable and which because of that property was named *R. diaporica*. Study of this organism in the laboratory led to human infection. Resemblance of the disease to the Australian "Q" fever and comparison of the strains showed that they were similar or identical. It is of interest to note that Noguchi probably isolated this organism in 1929 and was able to propagate it on leptospira medium for a few passages. Apart from the outbreaks at the institutes studying the organism little work was done at that time on the disease in the U.S.A. The Australian workers, however, continued their investigations and studied the cow as a source of human infection.

During the 1939-45 war the Germans noted the occurrence of broncho-pneumonia in their troops, principally in the Balkan countries, and named the disease "Balkan-grippe" because of its geographical relationship and its clinical resemblance to influenza. They noted that the natives in these regions did not become affected and that the returning German troops apparently did not convey the infection home with them. They isolated the infective agent in experimental animals but did not identify it. At the end of the war a number of outbreaks of non-specific pneumonia in Allied troops which occurred in Italy were identified as "Q" fever.

At about the same time a number of spectacular outbreaks of disease in U.S.A. were identified as "Q" fever and a series of investigations in California showed the widespread occurrence of infection amongst cattle, sheep and goats in that State. Investigation of the human population also showed widespread infection. Further examination of these human infections revealed three sources of infection:—

(1) Occupation in livestock or dairy industries.
(2) Residence close to livestock or dairies.
(3) Household use of raw milk.

About half of the infections could not be accounted for. This failure to trace the source of infection has been a prominent feature of many "Q" fever enquiries.

It is now known that the disease is world wide. Two important discoveries facilitated progress; Cox (1941) showed that the rickettsiae grew abundantly in the yolk sac of chick embryos, whilst by Craigie's ethyl ether extraction process the rickettsiae could be prepared in a highly purified form. So far as is known the antigen is specific and the antibody can be recognised readily in serum from the host.

R. burneti has a wide range of hosts. As stated earlier, ticks are infected, and the number of species reported from various parts of the world is large. At one time ticks were thought to be principal transmitters of infection but infection is now believed to be mainly by inhalation. Ticks have resumed some of their importance by the surprising discovery by Hengel, Kausche and Sheris (1950) that infected ticks can be brought in by dogs. In a village epidemic in Germany they were unable to find a satisfactory source of the infection. In the course of the investigation they freed dogs from ticks and sent them out into the fields. On their return they removed and tested the ticks collected by the dogs and found them positive for *R. burneti*. Giroud has shown that the louse can also become infected with *R. burneti* but the infection kills the louse in a few days.

Amongst the higher animals infection has been demonstrated in cattle, including the zebu, in sheep, goats, pigs and dogs and the bandicoot. It is likely that wider search would show more species infected, though some are probably immune, like the rabbit.

Except for two workers, it seems to be generally agreed that *R. burneti* causes no disturbance to its hosts among the domestic animals. Caminopetros (1948) maintains that in Greece the organism causes broncho-pneumonia in sheep and goats. Kilchsperger (1949) has found it associated with abortion in goats in Switzerland.

The great importance of the domestic animals as a source of infection to man is the large quantity of the organism which is excreted. In lactating animals it is present in the

unaltered milk whilst at parturition the foetal membranes may be heavily infected. With sheep and guinea-pigs it has been shown that infected milk readily infects the young of these species. *R. burneti* stains similarly to *Br. abortus* and to the organism of enzootic abortion in sheep and its presence in the foetal membranes may cause confusion in diagnosis.

R. burneti has great resistance to drying and to physical agents and is also unusually resistant to disinfectants. This uncommon combination of properties and the production of the organism in large quantities by infected hosts naturally facilitate spread. Human infection by inhalation can arise from the dust in infected animal buildings, from handling and processing wool and hair and from milk by contact or by ingestion. Experimentally, *R. burneti* has been recovered from the air of the buildings in which infected sheep were kept (Lennette & Welsh, 1951).

"Q" Fever in the Human Subject

"Q" fever is an acute febrile disease with pneumonia and some involvement of the central nervous system. Characteristically, onset is sudden after an incubation period of 14 to 26 days, with a sharp rise in temperature and generally a severe headache which is most aptly described in German as "quälender Kopfschmerz." Inapparent infection is quite common and there are all grades, from this inapparent infection to severe typhus-like disease. There may be well-marked lung lesions visible by X-ray in clinically inapparent infection. The disease is usually more severe with increasing age. Duration varies from a few days to several months. During the febrile stage the organism is present in the blood stream and is excreted in the urine and sputum. Spread from human to human is apparently uncommon but Siegert, Simrock and Stroeder (1950) have described a case in which a human being was responsible for an extensive outbreak in a hospital. The curious feature was that although *R. burneti* was easily demonstrated by animal inoculation in his sputum for five months he was infectious for his fellow-patients for only the two months during which his temperature was above normal. Inapparent human infection may of course be the source of vast quantities of infection amongst animals in contact. Fortunately in man the course of the disease is short and immunity following infection apparently is quite solid.

Many human infections have resulted from experimental work with the organism. No such work should be undertaken without adequate equipment and premises.

Some of the attempts to trace sources of infection in man with the knowledge at present available tend to have a fairy-tale quality. Nevertheless, some remarkable outbreaks have been described. An outbreak in a slaughterhouse described by Shepard (1947) illustrates well some of the startling features of these outbreaks. Fifteen out of 19 people engaged in the sheep and calf killing and dressing floor of a large packing house in Chicago became infected over a short period. The operations are highly specialised, each man doing a small part of the preparation. Some of these men must have come in contact with millions of animals and as immunity to "Q" fever is probably absolute and life-long this must have been their first contact with the infection. The extraordinary feature of this and similar outbreaks is the short duration of the outbreak although the organism is so resistant. The infective dose may be very large and infection occur only when this large amount is available. These slaughterhouse cases are not always simple. Meyer and Eddie (1949) showed what difficulties may arise when examining samples from slaughterhouse workers who may have both "Q" fever and brucellosis. They recommend blood cultures and animal inoculations to give a clear diagnosis. Large-scale outbreaks will have all available resources applied to arrive at a diagnosis but sporadic cases probably escape diagnosis. It will be seen from the map that infection in this country is fairly widespread in cattle and may be equally widespread in the human being. It is too early yet to say that cattle are necessarily the source of human infection: they may each pick up infection in an infected environment. We have not yet been able to survey other species but the distribution is probably similar.

"Q" Fever in Domestic Animals

With the exception of Caminopetros and Kilschsperger no reports of any disease in animals caused by *R. burneti* have yet been made. Most workers incline to the opposite view, that there is an astonishing lack of symptoms. Luoto and Huebner (1950), who first recorded the presence of *R. burneti* in the foetal membranes of cattle, found no evidence that the large number of organisms present interfered in any way with the nutrition of the foetus. Ormsbee injected cows via the teat canal with infected yolk sac with uninfected yolk sac as a control. The animals were young cows in the second and third months of their first lactation and were free from mastitis as judged by leucocyte counts and blood agar plates. After inoculation the milks were examined for butter-fat, pH changes, lactose, chloride, total nitrogen, casein, total solids, total ash. The blood haemoglobin was also measured. There was considerable disturbance caused by the injection of foreign matter but the injected quarters rapidly returned to normal.

The animal which has received most attention is the cow, followed closely by the sheep. Most work on artificial and natural infection has been done in California and that chiefly in the Los Angeles area. In Los Angeles the conditions are somewhat unusual with a dense population of milking cows in a kind of battery system, and low humidity. Infection in the cow is probably by inhalation and as conditions in that area for the production of large quantities of infected dust are optimal the infection rate for cattle is high. The infection rate for people living in the vicinity of these dairies is also high.

Cattle can easily be infected by the intramammary route but natural infection of the udder is probably a localisation of haematogenous infection (Stoenner, 1951). The occurrence of placental infection in the absence of infection in the bull (Huebner & Bell, 1951; Luoto & Huebner, 1950) is a little difficult to understand because although the bull may easily recover from the infection there should be antibodies as a relic of the infection. The marked predilection for the mammae and placenta in domestic animals should also hold good for the human female but has not so far been reported.

The heavily infected foetal membranes are probably the main source of infection for other animals and those in contact with such animals. There is a well-marked seasonal incidence of human infection from sheep associated with the lambing season while infection from cows is not seasonal. The marked difference in infection rates between abattoir workers handling few and many dairy animals was shown by Huebner and Bell (1951). Contact with infected milk is probably more dangerous than drinking it and the handling of a heavy udder oozing infected milk would account for many of the slaughterhouse infections. The infection of the tick is more difficult to understand.

Infection in the cow may persist for many months or

years. Infection rates in California are high but the data available here suggest that the infection rate is low and that spread is very slow. Treatment of an infected udder is probably ineffective and is certainly not economic. A great deal of work remains to be done on cattle but the evidence at present is that eradication is not very practicable.

Much more information on "Q" fever infection in animals is required. Some of the unidentified fevers in animals might repay investigation.

"Q" Fever in Great Britain

The first report of "Q" fever in this country was made by Stoker (1949). Three sera from 24 cases of human atypical pneumonia were positive for "Q" fever. At about the same time a number of people became infected in a hospital with a disease eventually identified as "Q" fever. Tracing of these cases revealed infection in cows in Kent and Devon (MacCallum *et al.*, 1949). Since then a number of human cases have been found in some of which the cattle with which the cases were associated were infected.

Early in 1950 it was decided to investigate the extent of infection in cattle in this country. This included the production of the antigen because it is both scarce and expensive and for reasons to be discussed elsewhere the available antigens were not altogether satisfactory. Two main sources of information were available:—

(1) The large number of cattle blood samples sent to the laboratory for serological, biochemical or other examination.

(2) The large number of guinea-pigs inoculated in various laboratories throughout the country for the examination of milk for the presence of the tubercle bacillus.

The test used is the haemolytic complement fixation test and the technique in use at Weybridge for "Q" fever is as follows: The final volume is 1·0 ml. in 75 by 10 mm. tubes. The amount of complement is 2·2 × 100 per cent. haemolytic units. The figure of 2·2 instead of 2·0 units was adopted to allow for deterioration of the complement during the course of manipulation and coupling of the complement with the large number of tests to be done. In fact no deterioration was found, but for uniformity and because of the difficulty of changing a test that has become routine the 2·2 units were retained. A little more than 1·0 unit of Henzerling antigen is used, the unit being the checkerboard unit. Sheep cells are 3 per cent. with 5 units of haemolytic antibody.

The procedure is to dilute the serum to 1 in 8, inactivate it at 60° C. for 30 minutes, separate into two tubes, add antigen and complement and store overnight at 4 to 6° C. Allow 15 minutes to warm up to room temperature, during which time the red cells and haemolytic antibody are left to couple, add this mixture to the tubes, incubate at 37·5 ± 0·1° C. for 15 minutes, and read after 30 minutes on the bench. The bath is so constructed that it will regain its working temperature within three minutes after insertion of a full load of tests. Positive controls and complement titrations are included in each day's tests. Any sample showing complete fixation or not more than a trace of haemolysis, that is, 4+ or 4− and not more than 1+ anti-complementary effect, is taken to titre.

The preparation of a suitable antigen gave difficulty principally because of slow delivery of equipment. Eventually, a satisfactory antigen was prepared in quantity and the source of the troublesome non-specific reactions identified as contaminating substances from the egg which could be removed by adequate washing of the antigen. A number of modifications were also made which increased the yield. Precautions prescribed for the handling and storage of "Q" fever antigens were found to be mainly unnecessary. Antigens could be frozen without harm.

With this antigen and with the fullest use of automatic syringes to measure the various reagents, and simplified recording and reporting methods, blood samples could be handled in large numbers. The sharp drop in reactions at the screening dilution of 1 in 8 with clean antigens meant that results could be obtained with the minimum of reagents and handling of cattle sera. As the survey proceeded the main effort was devoted to guinea-pig samples and although these samples were apparently independent of the purity of the antigens used, the number of cattle sera was still sufficient to cause serious delays if confusing reactions had occurred.

Table I shows the "Q" fever titres of guinea-pigs infected with milk under test for another infection, *viz.*, tuberculosis, and the titres of bovine sera.

When a bovine blood sample gave a reaction more than a trace, a sample of milk from the cow and from the herd was obtained if possible: Table II shows the results of biological tests and the titres. Not less than two guinea-pigs were used, which were killed at 30 to 42 days and the serum tested for antibodies.

A preliminary experiment with positive milk to which 5 per cent. boric acid was added to give a final concentration of 0·5 per cent. showed that at room temperature milk would remain infective for at least 54 days. The last test at 80 days failed because by this time the milk was so altered that it killed the guinea-pigs within a few minutes. These tests started in August and finished in November. All milk samples sent through the post were preserved with 0·5 per cent. boric acid.

There seems to be no correlation between blood titre and excretion of the rickettsiae in the milk. The low titre of the positive goat is surprising.

Table III shows the number of farms in each county from which biological and bovine samples have been obtained and the number positive for each. "Biological" means that a blood sample from a guinea-pig injected with milk from the farm was positive for antibodies to "Q" fever—

Table I
Titres of Cattle and Guinea-pig Sera

Species	Titre — 3 + at											Total tested*
	1/8	1/10	1/20	1/40	1/80	1/160	1/320	1/640	1/1,280	1/2,560	1/5,120	1/10,240	
Cattle	34	5	16	7	9	2	4	3	0	0	1	0	3,767
Guinea-pigs	21	11	29	39	70	55	38	19	8	0	1	0	8,343

*Totals are those tested with Weybridge antigen.

that is, if it had a titre of at least a trace at 1 in 40. In fact, this was unduly critical. On a number of occasions guinea-pigs with titres lower than this were later fully confirmed by a guinea-pig injected with the same sample but killed later, or by separate positive samples from the same farm or guinea-pig samples sent in by different laboratories. The extent of spontaneous "Q" fever infection in guinea-pigs in this country is not known but appears to be low or non-existent.

TABLE II*

Antigen	Serum titre	Biological result	Remarks
Commercial	4/20, T/40	—	Goats
,,	3/8	+	
,,	Bulk milk from goat herd	+	
,,	3/40, T/80	—	Cow, same farm
,,	3/8	—	Cow. Known "Q" fever herd
,,	2/20	—	Cow
,,	3/20	—	Cow
,,	Numerous 4/8 :		
	Herd (1)	—	Herd
	Herd (2)	—	Herd
Weybridge	4/40, 2/80	—	Cow
,,	3/20, 2/40	—	Cow
,,	3/40, T/80	—	Cows from same herd
,,	3/20, 2/40	—	
,,	3 at 3/8	—	
,,	4/40, 1/80		
	1/20	—	Bulk herd sample only
,,	3/40, T/80	—	Cow
,,	3/20, 2/40	—	Cows from same herd
,,	3/10	—	
,,	4/40, 4/80	—	
,,	2/10, 1/20	+	
,,	4/10, 2/20	—	
,,	Bulk milk from herd	—	
,,	4/320, 2/640	—	
,,	Bulk milk same herd	—	
,,	4/640	+	
,,	4/40, 1/80	+	
,,	4-/10, 4-/20, 1/40	+	
	Bulk milk from remainder of herd	—	
,,	4/40, 3/80, 2/160	—	
,,	Bulk milk from herd	—	
Commercial	4/320	+	
,,	4/640	+	
,,	4/320	+	
,,	4/20, 3/40	+	
Weybridge	4/640, 1/1,280	+	
,,	4/80, 4-/160	+	
,,	4-/40, 4-/80, 1/160	+	
,,	4/2,560, 3/5,120	+	

Figure to left of / is degree of fixation: figure to right of / is the dilution of serum.

4 = 100 per cent. fixation.
4- = Trace of haemolysis.
3 = 50 per cent. haemolysis.
2 = Between 3 and 1.
1 = Cells easily visible.
T = Trace of cells.
Thus 3/8 = 50 per cent. haemolysis at a dilution of serum of 1 in 8.

* It is regretted that, through a printer's error, the lines in the above table were omitted from the paper as circulated.

The results are weighted by a number of factors. There is always a tendency for more bovine samples to come from the better class self-contained herd whereas the guinea-pig samples will tend to come mainly from "flying" herds. Herds containing tuberculous cows obviously require repeated sampling.

Samples from one large dairy plant showed clearly the efficacy of pasteurisation. Samples taken for efficacy of destruction of tubercle bacilli showed that *R. burneti* was also destroyed. Tests at this plant also showed that *R. burneti* could quite easily be recovered from 3,000-gallon tankers.

Table IV shows the total farms and percentages for England, Wales and Scotland. In the areas tested in England there is the considerable percentage of 6·9 infected with *R. burneti*.

TABLE III

FARMS IN GREAT BRITAIN TESTED FOR "Q" FEVER TO JUNE 19TH, 1952

County	Biological samples	Biological positives	Bovine samples	Bovine positives
ENGLAND				
Bedford	4	—	—	—
Berkshire ...	18	—	—	—
Buckingham ...	51	2	—	—
Cambridge ...	10	—	—	—
Cheshire	149	20	1	—
Cornwall	4	1	78	1
Cumberland ...	4	—	—	—
Derby	54	5	—	—
Devon	108	2	281	3
Dorset	—	—	—	—
Durham	64	6	1	—
Essex	22	1	—	—
Gloucester ...	8	—	—	—
Hampshire ...	—	—	—	—
Hereford	9	2	—	—
Hertford	—	—	—	—
Huntingdon ...	4	1	—	—
Kent	—	—	—	—
Lancashire ...	75	8	—	—
Leicester	39	5	—	—
Lincoln	31	—	7	—
Middlesex ...	1	—	—	—
Norfolk	41	—	2	—
Northampton ...	31	6	—	—
Northumberland	1	—	—	—
Nottingham ...	111	1	2	1
Oxford	21	1	—	—
Rutland	1	—	—	—
Shropshire ...	148	6	—	—
Somerset	94	2	—	—
Stafford	427	40	—	—
Suffolk	2	—	1	—
Surrey	3	3	—	—
Sussex	244	10	—	—
Warwick	305	21	—	—
Westmorland ...	—	—	—	—
Wiltshire	1	—	—	—
Worcester ...	23	—	—	—
Yorkshire ...	473	36	—	—
Total	2,581	179	373	5

Table III continued on page 750

Q Fever
Location of Farms Tested
Each dot represents one farm
Orkney Islands

Q Fever
Location of Positive Farms
Each dot represents one farm

TABLE III (*continued*)

County	Biological samples	Biological positives	Bovine samples	Bovine positives
WALES				
Anglesey	50	—	4	—
Brecon	9	1	19	1
Caernarvon ...	107	—	8	—
Cardigan	4	1	29	1
Carmarthen ...	53	3	5	—
Denbigh	3	—	17	4
Flint	7	—	5	1
Glamorgan ...	259	3	5	—
Merioneth ...	—	—	—	—
Monmouth ...	41	2	3	—
Montgomery ...	1	1	22	—
Pembroke ...	19	—	5	—
Radnor	—	—	9	—
Total	553	11	131	7
SCOTLAND				
Aberdeen ...	111	2	—	—
Angus	—	—	—	—
Argyll	—	—	1	—
Ayrshire	21	—	1	—
Banff	16	—	—	—
Berwick	—	—	—	—
Bute	—	—	—	—
Caithness ...	—	—	—	—
Clackmannan ...	—	—	—	—
Dumfries ...	1	—	—	—
Dunbarton ...	—	—	—	—
East Lothian ...	—	—	—	—
Fife	—	—	1	—
Inverness ...	1	—	—	—
Kincardine ...	42	—	—	—
Kinross	—	—	—	—
Kirkcudbright ...	—	—	—	—
Lanark	—	—	1	—
Midlothian ...	8	—	—	—
Moray	7	—	—	—
Nairn	—	—	—	—
Orkney	32	—	—	—
Peebles	1	—	—	—
Perth	—	—	—	—
Renfrew	—	—	1	—
Ross	—	—	—	—
Roxburgh ...	—	—	—	—
Selkirk	—	—	—	—
Stirling	—	—	—	—
Sutherland ...	—	—	—	—
West Lothian ...	—	—	—	—
Wigtown	—	—	—	—
Total	240	2	5	—
England	2,581	179	373	5
Wales	553	11	131	7
Scotland	240	2	5	—
Total	3,374	192	509	12

TABLE IV
FARMS INFECTED WITH "Q" FEVER

England	179	6·9 per cent. of farms tested.
Wales	11	2·0 per cent. of farms tested.
Scotland	2	0·8 per cent. of farms tested.

SUMMARY

Blood samples from 6,227 cattle and 8,589 guinea-pigs have been examined by the complement fixation test for antibodies to *R. burneti*. Because of small differences in antigen these figures must be reduced to 3,767 cattle and 8,343 guinea-pigs. If a 3+ reaction at 1 in 8 and above is taken as significant, as in Table I, 2·1 per cent. of cattle have antibodies to *R. burneti*. The guinea-pigs samples represented 2,581 farms in England, 553 in Wales and 240 in Scotland, giving 6·9 per cent. infected farms in England, 2 per cent. in Wales and 0·8 per cent. in Scotland. There are gaps in the survey but sufficient samples were obtained to show the frequency and distribution of the infection in cattle.

It is hoped to survey other species in future.

Acknowledgments.—Thanks are due to the laboratories of the Veterinary Investigation Service and of the Public Health Laboratory Service without whose co-operation the survey could not have been made. The preparation, listing and the dispatch of the samples represented a great deal of extra work for them.

I am indebted to Mr. E. E. A. Ellerby and to Mr. B. Blowers for technical assistance in this survey.

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The CHAIRMAN: Thank you, Dr. Slavin. It is with very great pleasure that I introduce to you now Dr. Findlay, who is Director of the Public Health Laboratory Service at Wakefield. Dr. Findlay is a medical man who has for many years been interested in rickettsia and has shown quite a deep interest in this particular subject, "Q" fever. He is here really to give us the human angle on this disease which, from what we have heard, is perhaps more important than the animal angle. (Applause.)

Discussion

The discussion was opened, as follows, by Dr. H. T. FINDLAY, of the Public Health Laboratory Service, Wakefield: Dr. Slavin has shown us that the *potential* source of infection for man in the cattle of this country is quite considerable; 6·9 per cent. of farms tested showed evidence of infection.

What we are particularly interested to know is how much infection is actually passed to man and the manner in which it is passed, and of what significance "Q" fever is in this country.

First, as regards animals. As we have heard, most workers are agreed that infection in cattle produces no apparent ill effect either on the animal or production of milk in a dairy herd.

I suppose it is possible, nevertheless, that such a herd might be more susceptible to other infection—tubercle or brucellosis.

Just to diverge for a moment on the claim of Caminopetros in Greece that *R. burneti* produces broncho-pneumonia in sheep. Typhus rickettsiae notoriously affect debilitated individuals out of all proportion to their effect on those in better condition. This might account for his findings if the animals are ill nourished and in poor condition.

As regards human infections, I am indebted to Dr. Stoker (who has done so much on this subject) for my information. He tells me that he, and others, have records up to the present time of a total of 117 human infections of which 48 occurred in three epidemics and the remaining 69 as sporadic cases. He suggested that I should emphasise that although inapparent infections do occur (there is some ground for putting the figure about 3 to 1) clinical effects can be quite severe.

Between 1948 and 1952, so far as one knows, there have been only three epidemics of "Q" fever. We therefore do not seem to be prone to *frequent* outbreaks of clinical infection but we must remember that serious cases do occur and also that *R. burneti* may be the cause of much indeterminate ill health.

Dr. Slavin notes the short duration of these outbreaks in spite of the fact that we are dealing with an organism which is so resistant. This may be due to:—

(1) The great infrequency of case to case infection.

(2) The high degree of immunity.

(3) The sudden cessation of some infection, possibly aerosol, which rapidly disperses.

Diagnosis.—As we know, the diagnosis, particularly of sporadic cases, is a laboratory one—not only that but rather a specialised one.

1. It is a pity that *R. burneti* does not stimulate the production of agglutins to the proteus group—at least not to OX19 or OXK.

Would a study of proteus strains other than X19 and XK by those who have access to positive "Q" fever sera not be worth while? Laboratory diagnosis would be simplified and all laboratories could include a Weil Felix test in the routine examination of sera from P.U.O. cases in addition to enteric and brucella groups, Paul Bunnell test, etc.

2. There seems good reason to be satisfied on the specificity of the antibody tests as there appears to be little or no common antigenic relationship with other virus diseases. The only doubt I have seen cast on the complement fixation test was by Strauss and Sulkin, who found a significant correlation between a positive or doubtful Wassermann test and reactivity in the complement fixation test with *R. burneti*. Possibly, as Dr. Slavin found in his early work, their antigen was not truly specific owing to extraneous matter.

R. burneti is reported to cause an anamnestic reaction in T.A.B agglutinins—this is a point of some importance in these days of national service which would repay further observation. It certainly could cause confusion in the laboratory diagnosis of some of the typhus group fevers in India unless forewarned.

What is the reason for the apparent *absence* of complement fixing antibodies *in bulls* which Dr. Slavin and others have noted? This certainly does not apply to human beings, *e.g.*, in the Canterbury Art School outbreak the proportion of males infected was 80 per cent. though the school proportion was only 60 per cent. Have any active immunisation experiments been carried out in bulls to note their ability to produce antibody? This might not give all the information we require because it is possible they might produce antibodies on artificial immunisation but not on natural infection. Marked predilection for the mammae and placenta has not so far been noted in the human female (as Dr. Slavin observes) nor have females had consistently higher antibody titres as might be the case if the mammae were the optimum site for antibody production.

There is good correlation between excretion of rickettsiae in milk and the antibody titre in milk though not the same degree between rickettsiae in milk and the blood titre. Is this in any way connected with the question we have just mentioned of mammae as a site of antibody production?

The coincident high antibody titre in milk with a high degree of infectivity (as occurs also in brucella infection) in itself seems an anomaly unless there is no correlation between agglutinins, complement fixing antibody and protective effect.

The detection of "Q" fever antibodies by the antiglobulin sensitisation or Coombs test has been studied by Coombs with promising results and more recently in whey by Marmion and Stoker. The increased sensitivity is a matter of importance in examining bulked samples of milk and materially assists a survey.

Epidemiology.—As Dr. Slavin has told us, the outstanding characteristics of *R. burneti* are its great resistance to drying, physical agents and disinfectants, coupled with the enormous quantities of the organisms that can be excreted from apparently healthy animals in various ways.

What we are most interested in, is the *usual* method or methods of spread in this country because the method of spread can obviously vary considerably according to local geographical and biological conditions. We know that infection of man, as of animals, can arise by the respiratory tract and possibly also by ingestion though this does not necessarily mean that these are the only channels of infection.

Epidemic, murine and scrub typhus can all be propagated by aerosol infection as we know only too well from laboratory infections in the past and yet their usual vectors are the louse, flea and mite.

In "Q" fever, however, there is good evidence to indicate that respiratory infection may be the usual method of infection. *Two at least* of the three epidemics so far reported in this country have been caused by respiratory infection and in the first one, as I have already mentioned, there was evidence of case to case spread from a patient with large numbers of rickettsiae in his lung which may have made him more than ordinarily infective.

I think the evidence of infection by ingestion is much less convincing. It has been advanced by some American writers (Huebner & Bell, 1951) that because specific antibodies occur 10 times more frequently in users of raw milk than in users of pasteurised milk this is evidence of infection by ingestion. This does not seem necessarily to be the case as there is still a possibility of spread by aerosol. I noticed that Dr. Slavin considers "contact with infected milk is probably more dangerous than drinking it," which I think is the correct view. I do not think we shall properly understand such problems until further study has been made of aerosol spread under various conditions—we cannot know anything about the infective dose, optimum particle size, site of lodgment in the lung, etc.

A study of aerosol spread requires proper facilities such as special premises and the special wind tunnel devised for such work by Druett and May. A spinning top sprayer is used as a source of homogeneous particles of pathogenic organisms. The dry particle size may be set at any figure between 1 and 20μ and the cloud concentration can be varied. Provision is made for humidity control. Animals may be exposed to the cloud, and samples taken from it.

In a letter to *Nature* Druett suggests that the infection or mortality among a batch of animals exposed to a given bacterial cloud depends upon the *total number* of organisms deposited in the respiratory tract of the host, *and not upon the time taken to deposit them*—provided, of course, that there is insufficient time for development of immunity. If this were equally true in the case of rickettsial infections there might be, as it were, a build up to an infective dose over

a number of days in an infected environment and might explain a number of infections not otherwise accounted for which is a feature of "Q" fever enquiries.

Control of Infection.—I suppose it is true to say that the ultimate control of "Q" fever among human beings in infected environments depends on the elimination or control of the sources in livestock but I should certainly hesitate to advance suggestions on therapeutics and prophylaxis in this company. I was, however, interested to note in a paper by Luoto, Winn and Huebner that although aureomycin is effective in treating "Q" fever in experimental animals and in man, it does not appear to be an effective agent for treating infected dairy cows. This is very much the same as Smadel found with chloromycetin; the antibiotic is rickettsiostatic and not ricketticidal. It is a very different thing treating cases and carriers.

I notice from Dr. Slavin's map that in spite of considerable testing Anglesey, Caernarvon and Glamorgan appear to be remarkably free from infection—is there any explanation for this? I was very interested to note his suggestion of a two-way traffic—not only that infection passed from animal to man but that "inapparent human infection might be the source of vast quantities of infection amongst animals." I rather doubt this because man to man infection even in the clinically ill is very rare—though it does occur—and also on the whole man has not quite the same opportunity for widespread dissemination.

Finally, I should like to say a word or two about pasteurisation of milk in the control of infection. Even if completely effective it will not, of course deal with other sources of spread nor will it deal with the risk of aerosol infection in those handling the milk before pasteurisation which may be quite considerable.

Marmion, B. P., MacCallum, F. O., and others found that treatment of naturally infected milk at 154° to 156° F. for 15 seconds was adequate to render the milk non infective for guinea-pigs and it was only with artificially infected milks that higher temperatures or longer times were needed. Lennette and others found that two out of 42 naturally infected milk specimens pasteurised by the H.T.S.T. method contained detectable amounts of rickettsiae, despite the fact that the milk had apparently been adequately pasteurised.

The question would seem to be how often in nature one is likely to encounter bulked or other samples of milk containing *R. burneti* in such concentration that they will resist normal pasteurisation procedures.

It would seem that it might occur now and again but it does not necessarily follow even then that such specimens would prove infective for man.

GENERAL DISCUSSION

The Chairman: Dr. Slavin and Dr. Findlay would now be very glad to answer any questions which members cared to put, but as time was rather against them members were asked to make contributions brief.

Dr. F. C. Minett: The subject of "Q" fever had come up in Turkey, where he had recently been privileged to spend about a year and a half. He and his colleagues had received an invitation from the World Health Organisation to take part in a survey of the animal population in certain parts of Turkey in order to determine what was really the incidence of the disease amongst cattle and sheep and goats. Without bothering the meeting with a lot of details concerning the number of cases found, the samples examined and so on, it could be said that this involved the examination of several hundreds of samples from cattle and rather fewer from sheep. The number of cases of rickettsial infection of "Q" fever found was very small indeed.

With regard to the procedure of testing, they received very specific instructions or suggestions from the W.H.O. authorities concerning the handling of the antigen, the method of conducting the test, and the interpretation of results and so on, and were warned particularly against the possibility of non-specific reactions occurring in low level dilutions. Particularly in the case of cattle, they were advised to do screen tests with a dilution of 1 in 16, according to a technique which rather differed from that laid down in Dr. Slavin's paper. They had got quite a few positives, especially in cattle, with that dilution. Any sera reacting were re-tested in higher dilutions, but they noted that in very nearly all those cases with a doubling of the dilution—1 in 32—or something rather beyond that, the reaction faded out.

They also had cases of positive reactions fading out after tubes had been allowed to stand on the bench. In further correspondence received from W.H.O. it was pointed out that that was probably due to a weak complement. Incidentally, very careful instructions had been given as to the testing of the complements and other reagents.

In a later communication from W.H.O. they had been warned that even a dilution of 1 in 16 could hardly be regarded as safe and were recommended to screen test at 1 in 32, because some false positives had been reported at 1 in 16 by people in America.

He was not quite sure how all this information applied to the work referred to by Dr. Slavin, but it did show that one had to be extremely careful in the interpretation of reactions.

It was necessary, he thought, if one was to determine the true incidence of "Q" fever amongst animals, to go into the matter far more thoroughly and to show, for example, that guinea-pigs which reacted were themselves immune to *R. burneti*, to show that there was a disease which could be passaged, and to note such things as pathological lesions. As Dr. Findlay had said, the matter was largely *sub judice* but one had to be extremely careful.

Dr. Slavin would probably say that the results obtained in Turkey—and presumably there were similar results found in other countries taking part in the survey—came about because the antigen that was used was at fault. That suggestion, if made, Dr. Minett said, he would find difficult to accept, because he thought an organisation of the repute of W.H.O. would be most unlikely to distribute an antigen which was not pure and had not been well standardised. The conclusion about the whole thing was, of course, that far more exact information was required about the distribution of the disease amongst animals in this country and other countries.

Dr. G. Slavin: The reason for adhering to the Weybridge test was to avoid non-specific reactions. If insufficient speed was used when centrifuging the antigen during preparation much of the rickettsiae remained in the supernatant and was discarded. Naturally there was reluctance to discard valuable antigen and the result was that egg material remained in it. The way to ensure that everything was included was to use very high speeds, but in that case, everything meant literally everything and some substance—not identified but deriving from the egg—went down with the antigen. The important point was—and he hoped to publish this later—that there was a critical amount of "g" to apply to the antigen. Excessive "g" resulted in non-specific factors. About 12 000 × "g" was used in preparing Weybridge antigen. If this was pushed up to 20.000 × "g," false reactions began to creep in. It was possible to centrifuge antigen giving non-specific reaction and obtain non-specific fixation with the supernatant with sera which would not give fixation with the rickettsial deposit from the same antigen. These sera obviously did not contain true antibodies to *R. burneti*. With the large number of tests now being done non-specific reactions would wreck the survey.

Dr. H. T. Findlay: Stoker and others had carried out several tests in addition to the haemolytic complement fixation test for determining the titre of antibodies to *R. burneti*. Coombs antiglobulin sensitisation test appeared to be the most sensitive and specific of these tests. If there was any reason to doubt the specificity of the antigen being used in the haemolytic complement fixation test, one might resort to the antiglobulin sensitisation test. Technically, it was more difficult and laborious to carry out but it would serve as a useful special control under the circumstances.

Professor C. A. McGaughey: He had nothing to add to this discussion from his own personal experience but he wanted to convey to Dr. Slavin and Dr. Findlay a few excerpts from a couple of papers published by Dr. Schmid and his co-workers in the *Ceylon Journal of Medical Science* on recent work in Ceylon. That work had been only preliminary and evidently a great deal more had to be done. The contributions that had been made by Dr. Slavin and Dr. Findlay would prove extremely useful in stimulating the Ceylon workers. The figures given by Schmid were:—

Of 151 slaughterhouse workers tested by the standard W.H.O. technique (with which Schmid, an extremely meticulous worker, seemed to be quite satisfied) 16 had a positive titre of 1:16, one 1:32, one 1:64 and one 1:256; almost 4 per cent. of the slaughterhouse workers had positive titres. Of 100 buffaloes, two had a titre of 1:16. Of 100 meat cattle, two had a titre of 1:32. Of 104 goats, five had a titre of 1:32, two had a titre of 1:64, and one a titre of 1:256 (7·69 per cent.). Of 100 pigs there were no reactors. Of 102 sheep, two had titres of above 1:16.

In addition to the slaughterhouse workers, Schmid and his co-workers had gone to test 427 people who were more remotely connected with animals for example, men working in tanneries and leather factories and cold stores and butter and milk and meat departments, and they were all negative. Then 1,164 samples of human sera, which were negative for the usual salmonella, were tested. Twenty-one had titres of from 1:16 to 1:1,024, *i.e.*, 1·8 per cent. of cases of pyrexia of unknown origin were positive for "Q" fever bodies.

It had been found that many more sera were positive for "Q" fever than there were for typhus, and in Schmid's opinion "Q" fever was at least as important as typhus in Ceylon.

An interesting point was that in the positive cases among the slaughterhouse workers the medical histories were very vague. Practically none of them remembered any severe headaches or a definite

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CLINICAL COMMUNICATION

A CASE OF CONGENITAL HYDROCEPHALUS WITH HYDROMYELIA IN A CALF

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The subject was a heifer calf, the progeny of an Ayrshire cow, whose abdomen had appeared so abnormally large during the later months of pregnancy that it was assumed that she was suffering from hydrops amnii. When the mother was due to calve, however, an examination *per vaginam* revealed the fact that she was carrying a monstrosity, and as normal delivery appeared impossible and she was in good condition she was sent to the abattoir. After slaughter the calf was removed, when it was found to exhibit marked abnormality, the most obvious being the hydrocephalus.

The case is of unusual interest because not only was the actual hydrocephalus very pronounced but it was accompanied by extensive hydromyelia. There was also a marked distortion of the spine and of the fore part of the trunk.

The cranium (Fig. 1) was enormous, measuring over 32 cm. in its transverse diameter between the temporal bones, and having a capacity of about 20 litres. The cavity, however, was almost empty when the specimen was received, for its wall, composed of the skin and the cranial bones in the form of thin plates, had been incised to liberate the vast quantity of fluid contained within. Only a thin, friable, membranous lining remained in a collapsed condition inside the cavity. Similarly the vertebral canal was very much enlarged. This was at the expense of the bodies of the vertebrae which were greatly reduced in size. The transverse diameter of the atlas measured about 50 mm. and the vertebral canal in the lumbar region was of a similar width. The canal was occupied by a thin membranous tube which had collapsed. From the lateral walls of the tube the spinal nerves were seen to leave as delicate strands. Fig. 2 shows the great size to which the intervertebral foramina had attained in the lumbar region. The largest was about 50 mm. in diameter. The meninges were projected through the foramina as prominent bulges into the roof of the abdominal

FIG. 1.—Antero-lateral view of head.

FIG. 2.

FIG. 2.—View of spine from right side.

FIG. 3.—Transverse section through tail.

FIG. 4.—Spine, ribs and pelvic bone seen from above.

FIG. 3.

FIG. 4.

"Q fever"

fever. Some of them spoke about chest colds they had had but there seemed to be nothing very definite or suggestive of an attack of "Q" fever. However, one typical case of "Q" fever had been encountered in the last year and was fairly thoroughly investigated. Schmid found the titre was 1:1,024 and the only source of infection that could be suggested was from a herd of buffaloes that were tethered in a paddock close to where the man worked in a tea kiosk.

The comparative absence of known cases of "Q" fever in the general population was rather interesting. One might have thought there would be many more cases among the European population and that they would be a little more susceptible. But it might be a question of the very poor nutrition of the Ceylonese working class and the fact that there was less risk for the wealthier classes of Europeans, who were of course much better fed. (Applause.)

The CHAIRMAN: It was unfortunate to have to bring the discussion to a close but the limited amount of time available had been used.

Before closing the meeting, he wished to propose a hearty vote of thanks to both Dr. Slavin and Dr. Findlay for so ably putting before them the facts about a subject which was only slightly known to them. They would now be a little more *au fait* with "Q" fever when they met it in their travels, though obviously, as Dr. Findlay had said, there was quite a lot more to learn about this subject.

The vote of thanks was carried by applause.

cavity. The tube extended far back into the tail, reaching the level of the 10th coccygeal vertebra. Fig. 3 shows a transverse section of the tail in the region of the sixth vertebra. The tube is seen overlying the body of the vertebra which it rivals in outline. The bony vertebral canal, of course, is incomplete dorsally in this region so that the tube could expand dorsally beneath the skin without causing atrophy of the vertebral bodies. Both in the cranium and in the vertebral canal the nervous tissue had disappeared almost entirely as a result of pressure atrophy caused by the overabundance of the cerebrospinal fluid contained within, leaving only a very thin layer of nervous tissue supported by the meninges to enclose the fluid. On the escape of the fluid the surrounding walls collapsed into the cavity.

Fig. 4 is a view of the spine seen from above, and it illustrates the reverse S-shaped torsion of the vertebral column. It appears as if the pressure of the fluid within the vertebral canal has caused the column to yield in those regions where its walls are weakest, *i.e.*, in the regions of the brachial and lumbar enlargements of the cord where the intervertebral foramina would normally be largest. The thorax was drawn over to the right side and was very compressed laterally. Its contents were much reduced in size, but otherwise they showed little noteworthy change. As a result of the distortion of the chest walls the forelimbs in the region of the shoulder and arm were abnormally developed. The left shoulder was particularly distorted, the scapula, reduced to a thin narrow plate, occupying a groove between the more anterior thoracic spines and the upper portions of the left ribs which had come to assume the form of an inverted U. The musculature here, and indeed throughout the body, appeared to be poorly developed or atrophied, the white fibrous tissue predominating over the muscle tissue. The viscera towards the front of the abdominal cavity were also compressed and somewhat distorted, but there appeared to be no malformation here which could seriously affect function.

In considering the aetiology of the condition one is reminded of the three general factors which may be concerned.

It is thought that hydrocephalus may result from an excessive production of cerebrospinal fluid by the ependymal cells of the choroid plexuses of the ventricles. There appears to be little direct evidence in support of this view, although it is feasible that an increase in the rate of production of the fluid might be associated with a hyperaemia due to, say, meningitis. But Courville (1945a), discussing the condition in the human infant, states that, as far as his experience goes, the variety of hydrocephalus alleged to be due to excess activity of the choroid plexus (with the possible partial exception of tuberculous meningitis) appears to be a myth. In this connection it is to be noted that the carcase of the mother exhibited no lesions of tuberculosis when it was examined at the abattoir ; in fact, the cow had for some time belonged to an attested herd.

A second possible cause is an impairment in the mechanism of absorption of the cerebrospinal fluid. Here again there are few clinical records available in support of this as a common aetiological factor of the naturally occurring condition. It seems reasonable to assume, however, that any condition which would cause a rise in pressure of the intracranial and spinal veins, such as an intracranial tumour or obstruction to the return of blood from the head, might interfere with the reabsorption of the fluid into the blood stream. In the present case the compression of the thoracic viscera obviously could act in this manner, but in the reported cases of hydrocephalus in the domestic animals the condition appears to be most commonly unassociated with such widespread deformity of the thorax, and it would seem more likely that the abnormal form of the thorax was a secondary effect of the excessive amount of cerebrospinal fluid within the cord rather than the cause of a primary hydrocephalus.

Obstruction to the circulation of the cerebrospinal fluid from the ventricles of the brain and the central canal of the cord to the subarachnoid space is another and perhaps the most common cause of hydrocephalus. Walshe (1952) states that in the human subject " in about a third of all cases of congenital hydrocephalus there is a developmental defect, usually an atresia or imperfect development of the aqueduct of Sylvius. In other cases the ventricular foramina of exit are blocked by inflammatory exudates or the space in the opening of the tentorium is similarly occluded. In the remainder a new growth effects a block somewhere in the cerebrospinal system."

In the literature describing cases of hydrocephalus which have occurred among the domestic animals it is generally assumed, although unfortunately without direct evidence, that the cause is usually to be found amongst this group of factors. In most of the reported cases the cranium has attracted attention owing to its enormous size, and, as in the present case, its contents have consisted almost entirely of fluid with little brain substance available for examination. The concomitant hydromyelia in this case, however, suggests that there was no interference with the flow of cerebrospinal fluid between the ventricles or between the fourth ventricle and the central canal of the cord, but that the fluid had failed to reach the subarachnoid space through the occlusion of the apertures in the roof of the fourth ventricle. What the nature of that occlusion was it is, of course, impossible to surmise.

Another aspect of the occurrence of congenital hydrocephalus which should be mentioned is that associated with the nutrition of the mother and foetus during the period of gestation. It is well known that malformations have been produced experimentally in the lower classes of vertebrates by subjecting the early embryo to the effects of certain chemical agents—Stockard (1910) carried out work in this sphere many years ago on fundulus ova—and it is often found that congenital hydrocephalus is associated with other deformities in the region of the head. Courville (1945b), again referring to the human infant, states that in 30,000 autopsies congenital hydrocephalus occurred 59 times, and he adds " congenital lesions of the brain are associated in about half the cases with congenital malformations elsewhere." The work of Richardson, de Mottier and Hogan (1946, 1947) suggests that some cases of congenital hydrocephalus may be attributed to factors associated with dietetic deficiency. These authors observed the occurrence of the condition in infant rats after feeding the mother on a diet which was deficient in some unidentified nutrient. It is true that not more than 2 per cent. of the total rats observed showed the condition, but this figure was so constant in the several groups of rats so treated that they felt justified in regarding diet as an important factor concerned with the development of this condition. They assumed from the fact that experimental and controlled animals were selected at random from the same stock colony that the abnormality had no correlation with genetic constitution. Although they were unable to ascertain the nature of the deficient nutrient it is significant that the diet fed to the rats whose progeny developed hydrocephalus differed from that fed to the rats which showed no deformities in their offspring by being deficient in 5 per cent. of a liver extract fraction or 1 per cent. of an eluate of a fuller's earth adsorbate of liver extract.

Finally, the important aspect of inheritance naturally comes to mind in discussing the aetiology of hydrocephalus. Unfortunately, little history of the ancestry of this subject is available. The mother belonged to a dealer. She had been

in his herd for about 14 months and had been served by his own bull. Her previous calf was perfectly normal, and there is no history of other progeny of either parent. Yet inheritance has been associated with the occurrence of hydrocephalus, and in this connection the work of Cole and Moore (1942) is of significance. These authors describe the occurrence of hydrocephalus (together with two other associated conditions which they term "assymetry" and "jumpy") in a Holstein-Friesian herd. Twenty-seven calves were bred from 15 cows, all being the result of sire-daughter matings. Five calves showed hydrocephalus whilst five were affected with the other conditions. From the data obtained they concluded that the sire carried a lethal gene responsible for the development of hydrocephalus and that it was a simple recessive in its mode of inheritance.

It would seem that the subject requires considerable further study from several aspects, and it is obvious, too, that the clinician who can provide important information regarding the previous history of the case can be of invaluable assistance in this study.

I wish to express my indebtedness to Mr. E. B. Jones, of Bangor, North Wales, for his kindness in sending me the carcase of the calf.

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———. (1945b.) *Ibid.* p. 68.
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——— & Hogan, A. G. (1946.) *J. Nutrit.* **32.** 459.
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Walshe, F. M. R. (1952.) "Diseases of the Nervous System." 7th edn. p. 308.

ABSTRACTS

Studies on the Agent of Canine Virus Hepatitis (Rubarth's Disease) *

I.—The complement fixation test was described by Rubarth who used saline liver extracts of infected dogs as antigens. The author has produced active purified antigens by absorption and elution from chick red cells ; but the best method was by precipitation with methanol at −30° C. Antigens prepared from liver and other tissues of dogs infected with virus hepatitis fixed complement with dog hyperimmune sera, whereas similar preparations from tissues of normal dogs and dogs suffering from acute liver necrosis of unknown aetiology failed to do so. Comparison of antigens and sera from dogs infected with British, Swedish and Australian strains of virus did not indicate any difference between them. Similar preparations from tissues containing para-distemper virus did not fix complement with sera from dogs recovering from canine hepatitis. Immune sera which fixed complement with methanol-precipitated antigens also gave a precipitin reaction with them, antibodies measured by CF and precipitin tests appearing in convalescent sera at the same time, 21 to 51 days after infection. No loss of antigenic activity, whether measured by CF or precipitin test, resulted from heating antigens to 56° C. for one hour, although 100° C. greatly reduced their activity.

II.—The authors infected dogs intraperitoneally, into the anterior chamber of the eye and intracerebrally, using two British, a Swedish and an Australian strain of virus. The type of kerato-conjunctivitis produced by inoculation into the eye and sometimes intraperitoneally was considered rather characteristic of the disease. With one British strain the disease produced in three dogs was mild, the incubation period of one being four weeks. The other British strain produced fatal disease in three dogs with typical lesions and intranuclear inclusions. The Australian and Swedish strains (two dogs each) produced mild disease manifested by kerato-conjunctivitis in three dogs, while the fourth showed evidence of infection only by the appearance of CF antibodies six weeks after infection. One of two kittens showed mild symptoms after intraocular inoculation and tissue extract from it produced infection in a dog. Tissue extract from a natural case three months after illness produced mild disease when inoculated intraocularly into another dog ; symptoms lasted from the ninth to the 16th day, antigen appearing in the blood at the fifth week and antibody after the seventh week. Pooled tissues from three other such dogs six months after illness failed to produce symptoms in a test dog, but antigen appeared during the fourth week and antibodies from the 10th week after inoculation. Lice from an affected dog fixed complement with immune serum and when inoculated into a test dog, antigen was recovered during the first, fourth and fifth weeks and antibodies from the 10th week. Eleven dogs which had recovered from natural or experimental infection were challenged. Only one showed any reaction while both controls reacted more severely than it.

Serological studies in this work led the authors to conclude that antigen might be present in the blood for short periods during eight weeks following inoculation and sometimes its presence coincided with the appearance of clinical signs. It is concluded that a recovered dog may harbour virus. Antibodies were not detected in acute, fatal cases. In less severe cases they first appeared 21 to 70 days after inoculation.

A. W. G.

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Virus (Psittacosis-related?) Causing a Pneumonia in Sheep *

The author records the successful isolation of an infective agent from the lungs of two out of five sheep dying of pneumonia, by the intranasal inoculation of affected lung tissue into Swiss mice. Appropriate staining of lung impression smears in affected mice and sheep revealed the presence of elementary bodies similar to those of the psittacosis-lymphogranuloma-pneumonitis group. The virus was readily propagated in the yolk sac of five- to six-day chick embryos and was capable of serial passage in mice, causing extensive pneumonia.

The intranasal inoculation of 15 c.c. of the supernatant fluid from a centrifugalised 10 per cent. suspension of infected yolk sac caused a sharp rise of temperature in a sheep (100·1° to 106·1°) followed in the next 23 days by an intermittent temperature of increasing frequency and magnitude associated with respiratory distress. At autopsy lesions of pneumonia were confined to the apex of both lungs from which the virus was recovered on mouse inoculation. Experiments established the absence of the virus in normal sheep and mice, and by means of the complement fixation test the virus was shown to be different from that of feline pneumonia but to be closely allied to that isolated by Stamp and McEwen and shown by them to be the cause of enzootic abortion in ewes. The absence of abortion in pneumonia-affected flocks

* **Studies on the Agent of Canine Virus Hepatitis (Rubarth's Disease). I.—Complement Fixation and Precipitin Tests.** Larin, N. M. (1951.) *J. Hyg. (Camb.).* **49.** 410–426 (9 refs.) ; **II.—The Pathology and Pathogenesis of the Experimental Disease Produced by Four Strains of Virus.** Parry, H. B., Larin, N. M., & Platt, H. (1951.) *Ibid.* **49.** 482–496 (13 refs.).

* **A Virus Possibly Related to the Psittacosis Lymphogranuloma Pneumonitis Group, Causing a Pneumonia in Sheep.** McKercher, D. G. (1952.) *Science.* **115.** 543–544.

and the inability of the abortion virus to cause pneumonia in sheep and mice is regarded as evidence against a common identity and a claim is made for the acceptance of the pneumonia virus as a new member of the psittacosis-lymphogranuloma-pneumonitis group of viruses.

W. T. R.

REVIEW

[Antibiotics. A Survey of their Properties and Uses. Pp. ix + 290, with 24 illustrations and numerous tables. London: The Pharmaceutical Press. 1952. 25s.]

The first edition of this book was issued in 1946 under the title of "Penicillin: Its Properties, Uses and Preparations." The success achieved by penicillin prompted many workers to search for other antibiotics and streptomycin was isolated from the culture of a soil organism. In addition to penicillin and streptomycin there are chloramphenicol, aureomycin, terramycin, neomycin and others. The book has been enlarged so as to include those newer antibiotics which are in general use. Their properties and uses are discussed under the heads: commercial manufacture, chemistry, stability, standards and methods of assay, experimental background, clinical applications, veterinary practice and pharmacy. A chapter deals with the legal aspects of antibiotics and a list of commercial preparations is given. The sections on the use of antibiotics in clinical and veterinary medicine, for which we are clearly indebted to Professor Kekwick, F.R.C.P., and to Dr. S. J. Edwards, F.R.C.V.S., respectively, are planned to provide easy reference to specific pathological conditions. Each chapter has a long list of references to original papers.

Chapter 8 is entitled "Veterinary Practice" and consideration is given to the sensitivity of bacteria of animal-disease-importance to the different antibiotics. There is a section on the blood concentration of the antibiotics and a similar one on their retention in the bovine udder. Specific diseases are then dealt with very briefly and those considered are: bovine mastitis, with scant attention to those other than the chronic form due to *Streptococcus agalactiae*; bovine pyelonephritis; actinomycosis; reproductive disease in cattle, which appears to be mainly associated with *Vibrio foetus* infection; swine erysipelas; canine leptospirosis and about 16 lines on infection due to *Mycobacteria*. This chapter consists of but 18 pages; this is perhaps all that could be spared for specific consideration of the veterinary uses of the antibiotics. It is true, nevertheless, as stated in the preface, that the requirements of veterinary surgeons and practitioners have been borne in mind as well as those of doctors and pharmacists. Many of the chapters which do not deal with the specific needs of veterinary surgeons will provide the practitioner with information on these products which he may have difficulty in finding elsewhere. The book should be extremely useful for the student of veterinary pharmacy.

There is a statement in the historical summary which could have been expressed differently if the author had cared to investigate the occupations of those concerned (see *The Veterinary Record*. (1950.) **62.** 734).

Weekly Wisdom

General Eisenhower's election as President, after one of the bitterest campaigns in American history, in the course of which he has suffered the full rigours of critical analysis, demonstrates what all through his career has been his outstanding characteristic from which much of his success would seem to spring—his ability, in good times and bad, in politics as in war, to inspire men's confidence. This is a human quality and bears little necessary relevance to political or military ability.—Washington Correspondent of *The Times*.

QUESTIONS IN PARLIAMENT

Foot-and-Mouth Disease (Proposed European Commission)

Mr. Hurd (November 13th) asked the Minister of Agriculture if Her Majesty's Government have now decided to take part in establishing a European Foot-and-Mouth Disease Commission under the auspices of the Food and Agriculture Organisation of the United Nations to operate an international plan for the control of foot-and-mouth disease in Europe.

Mr. Wells asked the Minister of Agriculture whether the United Kingdom will be represented upon the European Foot-and-Mouth Disease Commission.

Sir T. Dugdale: The proposal that a European Foot-and-Mouth Disease Commission should be appointed was made at a conference convened by the Food and Agriculture Organisation of the United Nations in Copenhagen during September and was strongly supported by the United Kingdom delegate. In accordance with the decisions of the conference, FAO have since been preparing drafts of an agreement for the establishment of the Commission and of a budget. These documents have not yet been received. They are to be discussed at a further international meeting early in December at which the United Kingdom will be represented. We support the principles of the appointment of a Commission with suitable functions and would hope to co-operate in the work.

New Slaughterhouses

Mr. Murray (November 10th) asked the Minister of Food how many new slaughterhouses are now being built of modern lay-out; where they are situated; how much capital is set aside for this purpose; and if he will give the number to be completed in 1952-53.

The Parliamentary Secretary to the Ministry of Food (Dr. Charles Hill): Nine new slaughterhouses, estimated to cost £1,208,000, are provided for in the present building programme: two are now in use and three will be completed in the current financial year.

I will, with permission, circulate a list in the *Official Report*.

Mr. Murray: Is the hon. Gentleman aware that that answer gives a bit of comfort to me at least, as some of the old, obsolete, out-of-date slaughterhouses have made me weep? I hope that the hon. Gentleman will push on this capital investment and get some of these out-of-date places put into reasonably good shape.

Sir T. Moore: Will my hon. Friend bear in mind that, despite their badness, there were 14,000 slaughterhouses before the war, and that there is at present tremendous congestion which leads to a lot of suffering among animals still awaiting slaughter in the yards? Will he therefore press on with the increase asked for by the hon. Member?

Dr. Hill: I would refer the hon. Member and my hon. and gallant Friend to the reply given by my right hon. and gallant Friend to a Written Question last Wednesday, in which future slaughterhouse policy was summarised. [See *Vet. Rec.* **64.** 740. —*Editor.*]

Mr. Hastings: May I ask when the hon. Gentleman anticipates that the present programme will be completed?

Dr. Hill: It is anticipated that the present programme of nine slaughterhouses, referred to in my answer, will be completed in 1954.

Following is the list:

Guildford, Fareham, Canterbury, Grimsby, Swindon, Salisbury, Sunderland, Bournemouth and Poole district and Bolton, Leigh and Wigan district.

The new premises at Guildford and Fareham are now in use and those at Canterbury, Grimsby and Swindon are expected to be ready for use by the end of next month. Work has not yet started on the other four slaughterhouses and they will not be ready for use before 1954.

Animal Experiments

Mr. Emrys Hughes (November 10th) asked the Minister of Supply for what purposes the scientists in his employ applied for licences for experiments on animals last year; and what animals were used.

Mr. Sandys: It would not be in the public interest to give this information.

Mr. Hughes: How can it be in the public interest to refuse to give a simple answer to this Question? Is it not a fact that the Minister will not give an answer because he is ashamed of these experiments on animals?

Mr. Sandys: The same answer was given repeatedly by the late Government to similar Questions and I see no reason to vary it.

Mr. HUGHES: On a point of order. In view of the unsatisfactory nature of the reply, I beg to give notice that I shall raise this matter on the Adjournment.

Mr. EMRYS HUGHES (November 11th) asked the Secretary of State for War for what purposes the scientists employed by his Department applied for licences for experiments on animals last year; and what animals were used.

Mr. HEAD: The only licence for which my Department applied last year was needed at the Royal Army Medical College, Millbank, in connection with the usual medical work of investigation and diagnosis of disease. The animals used were rabbits, guinea-pigs and mice.

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ATTESTED CATTLE, SCOTLAND

Mr. WELLS (November 11th) asked the Minister of Agriculture what percentage of the total cattle population are attested in Scotland.

Mr. NUGENT: Fifty-five per cent.

Mr. WELLS (November 13th) asked the Minister of Agriculture what percentage of the total cattle population are attested in Wales and in England respectively.

Sir T. DUGDALE: Fifty-eight per cent. in Wales; 30 per cent. in England.

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WILD BIRDS (PROTECTION)

Lieut.-Commander HUTCHISON (November 11th) asked the Secretary of State for Scotland whether he is yet in a position to introduce legislation for the protection of Scottish rare birds.

Mr. J. STUART: Proposals for strengthening and clarifying the existing law on the protection of wild birds have been submitted by the English and Scottish Wild Birds Advisory Committees. These proposals are being very carefully examined by my right hon. Friends but I regret that they cannot at present give any undertaking about new legislation on this subject.

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ANTHRAX, KENT

Mr. WELLS (November 13th) asked the Minister of Agriculture how many cases of anthrax have occurred in Kent during the 12 months ended October 31st, 1952, or the nearest convenient date.

Sir T. DUGDALE: Fifty-six cases have occurred in the 12 months ended October 31st, 1952.

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OLD HORSES (SALE)

Mr. PETER FREEMAN (November 12th) asked the Minister of Food if he will amend the terms of reference of the Committee on Horse Slaughtering to enable the Committee to enquire into the sale and resale of old horses before they go to the slaughterhouse.

Major LLOYD GEORGE: No, the Committee's terms of reference are sufficiently wide as they stand.

NOTES AND NEWS

Diary of Events

Nov. 26th.—Annual General Meeting of the North Wales Division, B.V.A., at Ruthin (Council Chamber), 2 p.m.

Nov. 28th.—Meeting of the Royal Counties Division, B.V.A., at Reading (Caversham Bridge Hotel), 2.30 p.m.

Dec. 3rd.—Meeting of the Lancashire Division, B.V.A., in the Veterinary Hospital, Liverpool, 2.30 p.m.

Dec. 4th.—Meeting of the Central Division, B.V.A., at the Royal Veterinary College, N.W.1, 6 p.m.

Dec. 4th.—Annual Dinner-Dance of the North of England Division, B.V.A., in aid of the V.V.B.F.

Dec. 9th.—Technical Development Committee, B.V.A. (to discuss Sheep Diseases Report), 11 a.m.

Dec. 10th.—Technical Development Committee, B.V.A. (Ordinary Meeting), 10 a.m.

Dec. 12th.—Winter General Meeting of the North of England Division, B.V.A., at King's College, Newcastle-upon-Tyne.

Dec. 12th.—Annual Dinner and Dance, North Wales Division, B.V.A., at Llandudno. (See Notice.)

Jan. 9th, 1953.—Dumfries and Galloway Division, B.V.A., Ladies' Guild: Annual Dance in aid of the V.V.B.F., at Dumfries (Cairndale Hotel). (See Notice.)

R.C.V.S. EXAMINATIONS

Membership Examinations.—Written: Thursday and Friday, December 4th and 5th, 1952. Orals and Practicals: Commencing Monday, December 8th, 1952.

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COUNCIL AND COMMITTEE MEETINGS OF THE B.V.A.

The following arrangements for the Council and Committee meetings of the B.V.A. in January, April and July of 1953, have been made by Council B.V.A.:—

January 14th, 15th and 16th in London.
April 29th, 30th and May 1st in Edinburgh.
July 29th, 30th and 31st in London.

The times of the various meetings will be published in due course.

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B.V.A. Congress, 1953

A Provisional Committee has been formed to organise this conference, to be held in Aberdeen in the week commencing September 6th, 1953. Mr. W. J. B. Robson, M.R.C.V.S., is Chairman of the Committee, and Mr. W. A. Greig, B.Sc., M.R.C.V.S., Local Secretary.

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The British Veterinary Codex (B. Vet. C.)

In order that the profession might be informed, the officers of the B.V.A. sought a statement from the Pharmaceutical Society as to the position regarding the preparation of the British Veterinary Codex, and the following is an outline of the position which has been reached:—

About two years ago the Pharmaceutical Society with the help and advice of the Royal College of Veterinary Surgeons and the British Veterinary Association appointed a committee to prepare a British Veterinary Codex. This Codex is intended as a book of standards for drugs used in veterinary medicine and as an authoritative reference book for veterinary surgeons and for pharmacists engaged in supplying medicines to veterinary surgeons. It is hoped that it will be found useful as a book of standards, not only in Great Britain but in the British Dominions and Colonies, and with this aim in view overseas corresponding members of the committee nominated by bodies affiliated to the B.V.A. have been appointed in various countries. The American V.M.A. has also nominated a corresponding member. The British Veterinary Codex is not intended as a text-book of veterinary medicine and no information will be given on diagnosis.

Style and Contents of the B. Vet. C.—A style similar to that of the British Pharmaceutical Codex will be used and the B. Vet. C. will consist of: Part 1: General monographs. Part 2: Antisera, vaccines and related products. Part 3: Formulary. Part 4: Appendixes.

General Monographs.—These will consist of monographs on chemical, vegetable and animal drugs used in veterinary medicine. The main title will be in English, with Latin and English synonyms as subsidiary titles. Detailed standards will be laid down for such substances as are not included in the current B.P. and B.P.C. A statement, which is intended to represent the opinion of an expert committee, will be made on the action and uses of the drugs in veterinary medicine; toxicity, sterilisation, storage and dosage, will all be considered. The provisional list of contents for this section comprises approximately 400 substances. Some 1,200 substances have been considered and the following broad principles have been adopted in deciding whether a drug is suitable for inclusion in the book:—

(1) All drugs with well-substantiated pharmacological activity and therapeutic usefulness to be included, provided satisfactory standards can be specified.

(2) Drugs in common use for which there is not indisputable evidence of their pharmacological activity, will be included if the clinical evidence of their value is considered strong enough.

(3) Wide use alone is not considered sufficient justification for inclusion.

Monographs on Antisera, Vaccines and Related Products.—There will be monographs on about 70 products of this type, including not only those used extensively in this country but many that are in use oversea. Each monograph in this section will include a brief description of the method of preparing the product, detailed standards and detailed information on storage, methods of treatment and dosage.

Formulary.—A section consisting of formulas for reputable veterinary preparations (compounded medicines) together with methods of preparation, standards and dosage.

Progress.—Various sub-committees have been formed and 340 monographs have been completed. It is estimated that if Parts 1 and 2 can be completed by the end of the year the B. Ver. C. should be published before the end of 1953.

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Control of Manufacture of Veterinary Therapeutic Substances

The following announcement, relating to the Diseases of Animals (Therapeutic Substances) Order, 1952, to come into operation on January 1st, 1954, prohibiting the manufacture and importation, except under licence, of certain veterinary therapeutic substances, such as vaccines and sera, has been made by the Ministry of Agriculture.

The Diseases of Animals Act gives the Minister of Agriculture and Fisheries power to prescribe standards of strength, quality and purity for certain veterinary therapeutic substances such as vaccines and sera, the purity and potency of which cannot be adequately tested by chemical means. These substances are widely used and are of great value in modern veterinary practice but because of their methods of manufacture and use they could be harmful if not prepared with the greatest care and fully tested before issue.

The therapeutic substances available in this country for veterinary use are generally of a satisfactory standard. The need has been felt, however, for official standards to be laid down and controlled as they are for the corresponding substances used in human medicine.

As a first step, the Minister of Agriculture and Fisheries has made an Order,* the Diseases of Animals (Therapeutic Substances) Order, 1952 (S.I. 1952 No. 1933), applicable to sera and antitoxins, vaccines and antigens. It prohibits the manufacture for sale or the importation of these substances except under licence and prescribes the standard of purity applicable to them and tests for determining whether that standard has been attained. The purpose of the Order is to secure that these substances are prepared in such a manner that they will be safe in use. It is intended, as soon as circumstances permit, to prescribe standards of strength.

The Order comes into operation on January 1st, 1954, thus allowing reasonable time for manufacturers to effect any necessary adjustments in their production arrangements. Until that date the Diseases of Animals (Importation of Therapeutic Substances) Order of 1936 will continue in force. Neither Order affects such import licensing arrangements as may be laid down at the time by the Board of Trade under the Import, Export and Customs Powers (Defence) Act, 1939.

Although the Order does not come into operation until 1954, manufacturers who intend to apply for licences are advised to communicate at an early date with the Secretary, Ministry of Agriculture and Fisheries, Animal Health Division, Hook Rise, Tolworth, Surbiton, Surrey. The Ministry will be prepared to advise them on the requirements of the Order and on any steps necessary to comply with the conditions attaching to licences.

* Copies may be purchased directly, price 9d., from H.M. Stationery Office at the following addresses: York House, Kingsway, London, W.C.2; 429, Oxford Street, London, W.1; 13a, Castle Street, Edinburgh, 2; 39, King Street, Manchester, 2; 2, Edmund Street, Birmingham, 3; 1, St. Andrew's Crescent, Cardiff; and Tower Lane, Bristol, 1; or by post (10½d.) from P.O. Box No. 569, London, S.E.1.

B.V.A. Insurance Scheme: Profits for Benevolence

Report was made to the General Purposes and Finance Committee at its last meeting that a cheque for £350 16s. 7d. had been received from Messrs. S. H. Cannon & Co., Ltd., being a proportion of the profit derived from the Accident and Sickness Insurance Scheme down to December 31st, 1950. The Committee decided that this money should be divided equally between the Victoria Veterinary Benevolent Fund and the National Veterinary Benevolent and Mutual Defence Society; in the latter case in aid of its benevolent work.

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PERSONAL

Appointment.—The Colonial Office announces the appointment of Mr. J. W. Macaulay, B.SC., M.R.C.V.S., D.V.S.M., Senior Veterinary Research Officer, Northern Rhodesia, as Senior Veterinary Research Officer, Kenya.

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Mr. Duncan A. MacPherson, B.SC., M.R.C.V.S., of Seale-Hayne Agricultural College, Newton Abbot, Devon, has been appointed to the Food and Agriculture Organisation of the United Nations and is now stationed in Afghanistan. Mr. MacPherson qualified at Edinburgh in 1927.

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Dr. W. M. Scott, Fryern Hall, Bridgwater, has had the honour of being elected a Knight Councillor of the Knights of the Round Table in addition to being already Knight Conestable and Master of the Horse.

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Births.—Brown.—On November 5th, 1952, at Grantham Hospital, to Dorothy, wife of T. C. Brown, M.R.C.V.S., a son—Michael Carr.

Moors.—On November 5th, 1952, at St. Katherine's Maternity Hospital, Southport, to Betty, wife of Wm. M. Moors, M.R.C.V.S., a daughter.

Williams.—On November 10th, 1952, to Mary, wife of Mr. Eric I. Williams, M.R.C.V.S., St. Clears, Carmarthen, a sister for Thomas and Michael.

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Forthcoming Marriage.—Bennet—Mapley.—The engagement is announced between James Robert Bennet, M.R.C.V.S., second son of Mr. and Mrs. T. P. Bennet, of Bearsden, Glasgow, and Daphne Maureen Mapley, elder daughter of Mr. and Mrs. S. Mapley, of Shepton Mallet, Somerset.

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Marriage.—Hartley—Bartlett.—On November 15th, 1952, at Altrincham, Cheshire, Derek Clifford Hartley, M.R.C.V.S., of 4, Motcombe Grove, Gatley, Cheshire, to Joan Doris Bartlett, of Hale, Cheshire.

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Death.—Their many friends in the profession will learn with deep regret of the loss sustained by Major Frank Hopkin, O.B.E., M.R.C.V.S., of 35, Manchester Road, Knutsford, Cheshire, in the death of his wife, Frances Shaw Hopkin, which took place on November 13th.

Major Hopkin, who retains the Treasurership, was for many years Hon. Secretary and Treasurer of the National Veterinary Medical and Mutual Defence Society.

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R.C.V.S. OBITUARY

Davis, Thomas N., M.R.C.V.S., 166, Guildford Avenue, Feltham, Middlesex. Graduated London, May 21st, 1891. Died November 6th, 1952; aged 91 years.

Pindar, Evelyn Stanley, M.R.C.V.S., Captain late R.A.V.C., 264, West Parade, Lincoln. Graduated London, July 17th, 1908. Died November 10th, 1952; aged 71 years.

Trewin, Frederick Thomas, M.R.C.V.S., 53, Abbotsham Road, Bideford, N. Devon. Graduated London, July 25th, 1896. Died November 7th, 1952; aged 79 years.

Wallace, William Rutherford, M.R.C.V.S., D.T.V.M. (Edin.), late Director of Veterinary Services, Malaya, of "Shrublands," Sutton Forest, N.S.W., Australia. Graduated Edinburgh, July 15th, 1921. Died suddenly November 8th, 1952.

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Mr. Frederick T. Trewin, M.R.C.V.S.

It is a penalty of retirement to the more remote parts even of these small islands that valued contacts are lost. So far as Frederick Trewin is concerned, however, for many his departure from Watford to Bideford emphatically did not mean "out of sight, out of mind." for they have retained a vivid recollection of this able veterinarian, who carried on a fine practice in the Hertfordshire town and district.

They will remember him, too, as a truly delightful person who was ever a welcome attender at Congresses of the N.V.M.A., and a loyal and very effective member of the Royal Counties and Central Divisions of the Association.

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Mr. William Rutherford Wallace, M.R.C.V.S.

Mr. William Rutherford Wallace, who was the son of the late Mr. and Mrs. J. C. Wallace, Aberdeen, was educated at Aberdeen Central School and Robert Gordon's College. He took his veterinary diploma in Edinburgh and for a time practised in Aberdeen as an assistant to the late Mr. R. E. Drennan. During the first world war he served in the Royal Army Veterinary Corps in France and Italy.

Twenty-five years ago he emigrated to Malaya, joining the Malayan Veterinary Services, and later became director of the services.

During the late war Mr. Wallace was a civil internee in Japanese hands and on release returned to his former post, retiring two years ago to Australia.

Mr. Wallace last visited Aberdeen in 1947 with his Australian wife. His brother, Mr. J. R. Wallace, lives at 11, Hilton Drive, Aberdeen.

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GENERAL OBITUARY

Mr. A. C. Doull, C.A.

Members of the veterinary profession and in particular the alumni of the Royal (Dick) Veterinary College will learn with regret of the sudden death on Saturday, November 8th, 1952, of Mr. A. C. Doull, C.A., at his home at 5, Lutton Place, Edinburgh, 8.

Mr. Doull held the post of Secretary and Treasurer of the College from October, 1923, until he, very reluctantly, had to retire owing to ill-health in February, 1948. In addition to his official duties at the College he will be well remembered for the very active part he took in all student functions.

Apart from his College life Mr. Doull had a wide variety of interests and this was made evident by the very large attendance, at the graveside, of friends and representatives of the many organisations and societies with which he was connected. The burial service took place at Grange Cemetery, Edinburgh, on Wednesday, November 12th, 1952, at 2.30 p.m.

* * * * *

THE LATE DR. J. T. EDWARDS

PERSONAL TRIBUTES

There are many who can pay tribute to J. T. Edwards far more adequately than I, yet particularly on behalf of the younger members of the profession, I must voice the gratitude that so many feel for his interest and encouragement in their endeavours for the profession. To any young member who showed the least promise of taking an active part in its political life "J. T." gave the utmost encouragement and spurred them on to greater efforts. He could always be relied upon to enter into a discussion on a tremendous variety of subjects; always his contributions were of value and he has often amazed by his versatility and scope. Many a chairman has had reason to be grateful to him for jumping into a discussion that threatened to flag, even if later it was necessary to bring the guillotine down on him—yet never were these contributions idly made, all contained information of value and of interest.

By those who are regular attenders at London meetings "J. T." will be remembered with affection and his familiar figure will be sadly missed. His warm ovation at the "Central" meeting the day before his death must have given him much pleasure and assured him of the esteem in which he was so widely held.

J. O. J.

. . . . . .

November 7th was a day which brought great sorrow to me, for it was then that I heard of the death of my mentor and friend, J. T. Edwards. It was as if a sun which had shone so brightly had suddenly gone below the horizon and left darkness. I retired to the quiet of my room, for the passing of such a man meant too much to my profession to be the subject of discussion as is our wont of lesser men.

My sense of personal loss was great but I realised that it was as naught to the calamity which had struck the profession so suddenly. J. T. was in the legitimate succession to the great men of the past. For many years he bestrode his contemporaries like a Colossus. His intellect, his scholarship, his erudition, his integrity, his fearlessness and above all his kindly spirit marked him as a leader and inspirer of men.

My first memory of him goes back to the days when he was assistant to Sir John M'Fadyean and it was his duty to give us instruction in protozoology and helminthology. He did not spare himself; his exposition was brilliant and it was plain to us that he was destined for future renown. This came his way, for no man had a greater right to the approbation of his fellows and his work brought untold benefit to mankind. His research and discoveries in rinderpest earned him a place amongst the immortals. It would be difficult to estimate the vastness of the benefit that his discovery brought to the world. We do know that its repercussions have vibrated throughout many lands to their economic benefit and not least it has led to a great reduction in animal suffering.

J. T. like all the truly great was a simple, modest and kind man. He spent his life immersed in his work and caring for others with little regard for his own comfort or pleasure. He venerated his teachers and the pioneers of the past. Like them he could work and produce results without the aid of elaborate premises or equipment. This is the hallmark of genius. He made an indelible impression on the world of science and I venture to prophesy that in times to come the name of Edwards will live and shine brightly when most of his contemporaries are forgotten.

His last contact with his professional brothers was at a meeting of the Central Veterinary Society and within an hour of his death. He had been very ill and when it was noticed that he had entered the room the members and their guests from the Society of Women Veterinary Surgeons demonstrated in no uncertain way their great joy that he was back amongst them. It is good to know that he passed peacefully beyond the veil and with the acclamations of his fellows ringing in his ears.

J. McC.

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Dr. J. T. Edwards's long and active association with the veterinary profession enabled him to appreciate clearly that changes in outlook and spheres of emphasis do occur with the advancement of knowledge. This led him to an endeavour to integrate veterinary science, past, present and future. He dug deeply into the annals of veterinary history and also back to Grecian science and philosophy. He made outstanding contributions to the science of his own time—notably his studies on methods of immunising cattle against rinderpest and on the influence of the ecological conditions of the hedgehog and the rat on their susceptibility to foot-and-mouth disease virus. Regarding the future, he was strongly of the opinion that the sciences of Animal Behaviour and Comparative Psychology would be more widely studied and would influence veterinary science. He served on the Council of the Association for the Study of Animal Behaviour and in a paper given to this Association in 1944 he again showed his appreciation of the value of the historical by giving a survey of the history of that discipline.

Despite his engrossment with his own work he still found time to take a keen interest in the work of others and to give them advice and encouragement. Anytime I wrote to him he always took a lot of trouble to give me at length the information required.

In his devotion to his profession it could be said he was "like unto a merchant man, seeking goodly pearls: who, when he had found one pearl of great price, went and sold all that he had and bought it."

A. B.

. . . . . .

Mr. R. H. Smythe, M.R.C.V.S., of Nottingham, writes:—

In the passing of Dr. J. T. Edwards, there departs from our profession one of its outstanding personalities of all time. Such men are rare, good without effort, great without a foe. Although his friends were everywhere, I rejoice in being able to claim a longer and perhaps closer acquaintance with the real man than many for, as a final year student, experimenting with one or two others in the matter of "digs" we accepted J. T. as one of our party in sharing the tenancy of a house. He was then a junior and in after years I often smiled to think that in those distant days he relied greatly on my experience whilst ever since, on countless occasions, I have been so dependent upon his. Even when away in foreign lands, J. T. never failed to write me voluminous letters in which he rarely omitted to refer with joyful reminiscence to "those good old days at 222."

Never was he so happy as when approached for advice and help. J. T. lived for his profession and for the welfare of his professional friends with never a thought of self. He was a gifted raconteur and a great speaker, but behind his measured phrases and majestic enunciation lay a lifetime of unremitting labour and earnest thought. I recollect that on one occasion he told me he considered he would be failing in his duty to his profession if he omitted to take an active part in every discussion. But, his was no empty speech; never have I encountered a subject, professional, scientific or political, with which J. T. was not completely conversant in all its details.

Tragic as was his ending, we may feel relieved that he was not condemned to a period of idle inactivity and suffering. To have lain helpless would have been unendurable torment to his active mind.

Great men like J. T. Edwards are never wholly lost to any profession. Always will he remain with us in spirit, an example to those who knew him and an inspiration to many generations to come.

. . . . . .

Professor C. W. Ottaway, of the Department of Veterinary Anatomy, The University, Bristol, writes: There must be many

voices and not a few pens anxious to pay tribute to the late Dr. J. T. Edwards at this time. In the light of his manifold activities and interests one could hardly think otherwise.

In humble respect to a great man I should like to touch on but two memories: his enthusiasm and skill as an historian and his encouragement for the pursuit of fundamental knowledge. A year or so ago it was my need to refer to some aspects of the history of our profession. I wrote to Edwards, and he replied at length, giving me fully of his vast knowledge, and in a covering letter there appeared the following typical comment:—

"If you do exactly what I tell you in the attached rough notes you will obtain readily, in a condensed form, all the information you need and you will not waste time uselessly."

To a young man it was a privilege to be able to say Edwards knew you, and since the day when I could first say that, some 15 years ago, I have always been deeply impressed by his insistence that the young research worker should get down to the root of the problem; superficial knowledge was not good enough. I am sure many will share those views.

Truly may we say as a profession—"Well done, thou good and faithful servant."

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The Funeral

The Rev. E. Wyn Evans, Minister of the Welsh Presbyterian Church, Charing Cross Road, conducted the funeral service at Golders Green Crematorium on the morning of Tuesday, November 11th. Though held at an early hour, a large company, representative of the manifold aspects of our calling with which Dr. Edwards had contact, was present to pay tribute. The following were family mourners: J. Michael Edwards, J. Griffith Edwards (sons); Mrs. Douglas Thomas (sister), Mr. and Mrs. W. George (brother-in-law and sister), Dr. and Mrs. S. J. Edwards (brother and sister-in-law); Mrs. C. M. Edwards; Dr. J. Picton Thomas, Mr. J. Wright, Mr. D. George (nephews).

Representing the Royal College of Veterinary Surgeons were Professors J. B. Buxton, J. McCunn and G. H. Wooldridge, Mr. W. G. R. Oates (Registrar); the British Veterinary Association: Mr. H. E. Bywater, Major H. Kirk and Mr. Frederick Knight (Secretary); Ministry of Agriculture: Mr. M. Crawford, Mr. J. R. Hudson and Dr. A. W. Stableforth, with Mr. W. A. Pool and Dr. E. L. Taylor; also Mr. H. H. Skinner, representing past colleagues at Pirbright; the Royal Veterinary College: Professor T. J. Bosworth (also representing the editors of the *Journal of Comparative Pathology and Therapeutics*) and Mr. H. W. J. Adams, Mr. Southeran (lay staff) and Mr. Dunn and Mr. Gray (students); the Agricultural Research Council: Dr. W. S. Gordon; Society of Women Veterinary Surgeons: Mrs. Hargreaves and Miss Joshua; the Society of Practising Veterinary Surgeons: Dr. Tom Hare; Herts and Beds Veterinary Society: Mr. S. B. Vine; the Veterinary Research Club: Dr. N. S. Barron. Dr. R. Lovell represented the Section of Comparative Medicine of the Royal Society of Medicine, Mr. H. W. Eley *The British Veterinary Journal*, Mr. W. Brown *The Veterinary Record*, and Mr. D. Tindall the publishing house of Messrs. Baillière, Tindall & Cox. Amongst many friends present were Mr. and Mrs. E. Brayley Reynolds. Mr. A. Brownlee, of Compton, and Mr. Thomas Lishman, with Mr. E. H. Knight, who was personal technical assistant to Dr. Edwards from 1929 to 1946.

On Friday, November 14th, a memorial service was held in the Old Church at Pembrey, S. Wales.

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ADDRESSES OF DISEASE-INFECTED PREMISES

The list given below indicates, first, the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

ANTHRAX:

Beds.—215, Common Road, Kensworth, Dunstable (Nov. 7th).

Berks.—Wheelright's Yard, Buckland, Faringdon (Nov. 8th); The White House, Mackrey, Brightwell cum Sotwell, Wallingford; Woodspeen Farm, Woodspeen, Newbury (Nov. 13th).

Bucks.—Litslade Farm, Newton Longville, Bletchley (Nov. 12th).

Cambs.—The Garden, Longview, Coton, Cambridge (Nov. 7th); New Barn Farm, Streatley End, West Wickham, Cambridge (Nov. 10th).

Ches.—Rainow Farm, Buglawton, Congleton (Nov. 10th); Love Lane Farm, Betchton, Sandbach (Nov. 12th).

Cornwall.—Killlow, Kea, Truro (Nov. 10th); Tremore Valley, Bodmin (Nov. 13th).

Derbys.—Bubnell Hall Farm, Baslow, Bakewell; Moorland View, Ashford Road, Bakewell (Nov. 10th).

Devons.—Lower Beara Farm, South Brent (Nov. 11th); Little Wadham Farm, Knowstone, South Molton; West Barton Farm, Molland Botreaux, South Molton (Nov. 12th); R.A.F. Station, Chivenor, Barnstaple (Nov. 13th).

Dorset.—Wood Acres, Gravel Hill, Merley, Canford, Wimborne (Nov. 10th); 167, Portland Road, Wyke Regis, Weymouth (Nov. 11th).

Durham.—Anesty Farm, Bishopton, Stockton-on-Tees (Nov. 10th).

Essex.—Baldwins Farm, Barling Magna, Southend-on-Sea (Nov. 7th); Suttons Farm, Barling Magna, Southend-on-Sea; Giffords Farm, Great Sampford, Saffron Walden (Nov. 11th); Resthaven, Eastbury Avenue, Hawkwell, Rochford; Bullocks Farm, Takeley, Bishop's Stortford (Nov. 13th).

Glos.—Lower Stone Farm, Lower Stone, Berkeley; Old Lodge Farm, Tortworth, Falfield (Nov. 11th).

Hants.—Overton Farm, Barrows Lane, Sway, Lymington (Nov. 10th); Westmark Farm, Steep, Petersfield; 26, Malvern Road, Moordown, Bournemouth; Grange Farm, Wellingborough (Nov. 11th); Cowdon Farm, Micheldever, Winchester; Deepdene Piggery, Dibden, Southampton (Nov. 13th).

Kent.—Piggeries, St. Radigunds Allotments, Dover (Nov. 8th); Atherfold, Ide Hill, Sevenoaks (Nov. 12th).

Lancs.—Pages Farm, Clitheroe; Brown House Farm, Wheelton, Chorley (Nov. 8th); Slaters Farm, Grimsargh, Preston (Nov. 11th).

Leics.—Sleath Farm, Gilmorton, Rugby (Nov. 11th).

Norfolk.—Crabtree Farm, Wood Dalling, Norwich (Nov. 7th); Breckland Farm, Larling, Norwich (Nov. 12th).

Somersets.—Lower Court Farm, Long Ashton, Bristol (Nov. 7th); Lower West Barn Farm, Witham Friary, Frome; Hillside Farm, Primrose Lane, Mudford, Yeovil (Nov. 8th); Parsonage Farm, Wembdon, Bridgwater (Nov. 10th); Smalldown Farm, Evercreech, Shepton Mallet; Jeffries Farm, New Road, East Huntspill, Highbridge (Nov. 11th).

Suffolk.—Francis Farm, Somerton, Bury St. Edmunds (Nov. 7th); Brunswick Farm, Kettleburgh, Woodbridge (Nov. 10th); Buttons Farm, Charsfield, Woodbridge (Nov. 13th).

Surrey.—Jordans Farm, Forest Green, Ockley, Dorking (Nov. 10th); Roppeleighs, West End, Haslemere (Nov. 13th).

Sussex.—Frogmore Farm, Plummers Plain, Horsham (Nov. 7th); Hayes Lane Pig Farm, Hayes Lane, Slinfold, Horsham (Nov. 8th); Springditch Piggeries, Steyning Road, Horsham-by-Sea; Woodsoames Farm, Rudgwick, Horsham (Nov. 10th).

Wilts.—Manor Farm, Alton Priors, Marlborough (Nov. 7th); Mill Farm, Manningford Abbots, Pewsey (Nov. 10th); Watergate Farm, Bulford Village, Salisbury; Dinton Field Barn, East Farm, Dinton, Salisbury (Nov. 12th); Paradise Farm, Christian Malford, Chippenham (Nov. 13th).

Worcs.—Pigeon House Farm, Leigh, Worcester (Nov. 8th).

Yorks.—Mayhills Dairy Farm, Silsden Moor, Silsden, Keighley (Nov. 10th); Oxenhurst Farm, Newton-in-Bowland, Clitheroe (Nov. 12th); Golton Bros., Kelfield, York; Gill Lead, Bentham, Lancaster (Nov. 13th).

FOWL PEST:

Sussex.—Posingford Farm, Hartfield, Tunbridge Wells (Nov. 12th).

SWINE FEVER:

Bucks.—Brackwell Farm, Lower Winchendon, Aylesbury (Nov. 10th).

Ches.—Landslow Green Piggeries, Hollingworth (Nov 12th).

Durham.—The King's Head Hotel, Barnard Castle (Nov. 10th).

Essex.—Rookery Farm, Nayland Road, West Bargholt, Colchester (Nov. 12th).

Herts.—Moor Farm, Sandy Lodge Road, Moor Park, Rickmansworth (Nov. 10th).

Lancs.—Jameson's Farm, Golborne Road, Ashton-in-Makerfield, Wigan (Nov. 11th).

Lincs.—Woldlime Ltd., Louth Road, Horncastle (Nov. 12th).

Norfolk.—Large's Premises, Lynn Road, Snettisham, King's Lynn (Nov. 10th).

Stirlings.—Auchenrooch, Milton of Campsie, Glasgow (Nov. 13th).

Wigtowns.—Piggery, Woodlands Croft, Barsalloch, Newton Stewart (Nov. 10th).

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UNIVERSITY COLLEGE OF NORTH WALES

Sir D. Emrys Evans, M.A., LITT., Principal of the University College of North Wales, Bangor, in a recent address to the Court of Governors at Bangor, said there was a substantial programme of research into problems relating to animal reproduction and nutrition being carried out in the departments of Zoology, Agriculture and Agricultural Chemistry. Most of this was supported by generous grants from the Agricultural Research Council. Taken together, they constituted a broad field of work on animal health that would give the college a strong claim to be considered as a suitable centre for a Welsh Veterinary College when circumstances allowed such an important development to be realised.

ROYAL COLLEGE OF VETERINARY SURGEONS

Animal Management Examination, November, 1952

List of Successful Candidates

London

Bedson, I. F.	Norman, D. A.
Bowles, Jeffrey	Sutton, J. B. (credit)
Finn, C. A.	Whitaker, D. L. (credit)
Gray, John	White, Frederick
Naylor, D. C. (credit)	Woollett, W. G. W. A.

Dublin

Dolan, T. C.	Lowndes, J. D.
Fleury, A. T.	McDonald, W. E.
Hernon, John	Stack, P. D. J.
Johnston, J. B. H.	York, A. J. (credit)
Kilroy, C. S.	

Edinburgh

Gray, Brian	Kirkbride, L. M.
Gripper, J. N.	Stewart, J. G.
Gyi, Ko Ko	Stokoe, W. M.

Pharmacology, etc., Examination, November, 1952

Dublin

Cox, Edward	Mackessy, E. J.
Deboys. D. C.	McManus, Dermot
Daly, D. G.	Noonan, J. D.
Dowdall, F. J.	Sheridan, J. J.
Fleming, A. R.	White, J. J.
McInerney, J. F.	

London

Abraham, B. F.	Jones, T. H.
Alexander, T. J. L.	King D. K.
Ansell. R. H.	Loving, Charles
Arnold, J. H. S.	McVoy, K. I.
Baldwin, I. J.	Maynard, H. M. F.
Barton, C. R. Q.	Mears, R. H.
Beddows A. C. L.	Nelson, G. J.
Beesley, C. J.	Nestel, B. L. (credit)
Benbow, G. M.	Olorenshaw, P. H.
Booker, J. C.	Skipsey, Roger
Breese, J. L.	Smith, Miss L. C.
Brocklesby, D. W.	Stalbow, Michael
Bywater, J. E. C.	Taffs. L. F.
Cansfield, C. J.	Thomas, C. J.
Cawthorne. W. N.	Thursby-Pelham, R. H. C.
Dommett. D. J.	Tiffin, R. B.
Gibson. F. E. T.	Vinson, M. A.
Hammond, Miss W. M.	Vinycomb. Miss H. M.
Hampshire. F. J. W.	Waller. J. F.
Holmes, J. R.	Walter. A. J.
Holmes, R. G.	Wells, J. R.

Liverpool

Shaib, Bukar

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

"WARFARIN" POISONING IN THE DOG

Sir,—In the *Record* of the 8th instant Mr. W. M. Taylor asks for information about "Warfarin."

Some time ago I suspected, but was unable to prove, that this material had poisoned two heifers. However, feeling that the increasing use of "Warfarin" might lead to deaths among domestic animals, I made some enquiries, and thanks to the help given me by Miss Raymer, Librarian, R.C.V.S., I can furnish a list of references:—

(1) Public Health Reports. Vol. 65. November 24th, 1950 (Federal Security Agency, Washington).
(2) Public Health Reports, Vol. 65. January and June, 1950.
(3) *Veterinary Medicine*. Vol. XLVII. No. 6. June, 1952.
(4) *Veterinary Medicine*. Vol. XLVII. No. 9. September, 1952.
(5) *Journal of the Royal Sanitary Institute*. March, 1952.

In addition, references to poisoning of cattle by damaged sweet clover will be found in the *Journal of the American Veterinary Association* (1923-24). Vol. LXIV. Sweet clover contains an anti-coagulant, "Coumarin." From the above, the following brief notes have been extracted: The Public Health Reports refer to the symptoms and *post-mortem* appearances of rats and other animals killed by "Warfarin."

"Warfarin" acts by inhibiting the formation of prothrombin and by causing capillary haemorrhage. The symptoms are pallor and weakness. Subcutaneous haemorrhage and/or swelling due to haematomata may become visible externally especially on the appendages. Oral and rectal bleeding occurs, but not in all rats. Appetite remains disproportionately good, although it does decrease. There are no convulsions or seizures. Secondary poisoning may occur if cats (and presumably dogs) eat several "Warfarin" poisoned rats over a period of days. *Veterinary Medicine*, Vol. XLVII, No. 9, September 1952, records the death of a number of pigs due to contamination of foodstuff with "Warfarin." One morning a number of pigs were castrated and by the afternoon of the same day many were dead, apparently as the result of haemorrhage, and others were bleeding from the operation wounds. Next day many gilts were dead or dying. Lesions demonstrated in the females indicated bleeding into the tissue spaces, principally on the inner surface of the front legs, while haemorrhages in the males usually involved the inner surface of the hind legs and groins. Whenever an animal was bumped by others in the pen, or a bruise was caused by straddling the troughs or by being trampled by the sow, large haemorrhagic areas and subcutaneous haematomata appeared. Bruises on the gilts appeared to result from straddling the troughs, while lesions on the boars apparently resulted from handling during castration. A few animals were bleeding from the nose and mouth, but none was passing blood by rectum. Haemorrhage and weakness were the only visible signs of disease. *Post-mortem* examination: Aside from the severe anaemia, the striking feature was the markedly prolonged coagulation and prothrombin times. Altogether, 56 out of 120 pigs died from accidental "Warfarin" poisoning through contamination of mill prepared food. The fatal cases occurred principally in those animals which had been castrated or bruised by rough handling.

There is an article in *Veterinary Medicine*, Vol. XLVII, No. 6, June, 1952, describing a series of "Deliberate Warfarin Poisonings." Altogether 61 dogs were poisoned. The owner of the "Warfarin Patent" issued a bulletin on the toxicity of this compound in which it is stated that 5 mg. per kg. of bodyweight, administered daily, would be fatal to dogs in from 5 to 15 days. Judging from the amount of poison recovered from the bait, the authors of the report concluded that each dog in the series must have ingested at least 1 to 3 gm. of the compound at one time. All of the dogs presented haemorrhage in some form, either in bloody vomitus and stools or in a final fulminating haemorrhage just before death. In addition, all animals showed cyanosed mucous membranes, rapid pulse, shock and subnormal temperatures. The *post-mortem* findings were: The general findings were severe petechial haemorrhage of the intestinal mucosa. In two of the dogs haemorrhages of the lungs were observed. There were no ulcerations of any kind in the digestive tract.

Forty-seven out of 54 dogs treated recovered. The treatment adopted was the intravenous injection of glucose saline supplemented with a vitamin K preparation (Koagamin), or glucose saline intravenously and vitamin K intramuscularly.

As a matter of interest, references to damaged sweet clover poisoning in cattle will be found in Udall's "Veterinary Medicine" and in the *Journal of the American Veterinary Medical Association*, Vol. LXIV, 1923-24. About 1,921 cases occurred of what appeared to be a new disease in cattle simulating haemorrhagic septicaemia and blackleg. The symptoms were of two types: An acute type in which the first indication was revealed by the carcase of a victim, or the animal was found with blanched mucous membranes and showing subcutaneous swellings. The animal appeared dull, standing quietly in its stall and still eating a little food until a short time before its death. Death in this type is almost always the direct result of haemorrhage. In many cases the haemorrhage is exclusively internal, no external evidence being apparent. Nature of the swellings: a typical swelling is not hot, contains no gas, is only slightly painful, and has no tendency either to slough or suppurate. These facts, taken in conjunction with the rapidity of their formation, indicate the non-inflammatory nature of the lesions. The swellings may either pit on pressure or fluctuate, depending on the rapidity with which they have formed. Most of the swellings contain either coagulated or uncoagulated blood. They vary greatly in size, the smallest being scarcely perceptible, while the largest may measure several feet in circumference, with an elevation of several inches. They may appear in any part of the body but occur with the greatest frequency on the back.

In the anaemic type the death-rate may be high but the disease is never so explosive as in the acute form. In this type occur deaths following de-horning, parturition and simple operations. The blood shows a delay in clotting time. Following operations they bleed to death.

In the article, Pammel is quoted to the effect that the sweet clovers

contain coumarin (C9H6O) which is found in the tonka bean, sweet vernal grass and vanilla grass. In Europe sweet clover is suspected of being poisonous . . . according to Friedberger and Frohner, sweet clover causes paralysis of muscles.—Yours faithfully, S. J. Motton, Penzance. *November 15th*, 1952.

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TREATMENT OF PREGNANCY TOXAEMIA IN EWES

Sir,—I was interested to read Mr. Griffith's communication on pregnancy toxaemia in ewes, particularly as I, too, have tried glycerol in the treatment of this condition.

Until this agent was employed I had found nothing of any use and had given up the idea of inducing abortion with stilboestrol as in my hands this treatment had been worthless. Having read of the treatment of acetonaemia in dairy cattle with glycerol I decided to try this agent for the relief of pregnancy toxaemia.

The dosage employed was 2 oz. twice daily for three days, and while results were not spectacular they were certainly encouraging.

Any ewe which was unable to lie on her brisket and keep her head up died in spite of treatment, but cases which were taken earlier almost always recovered.

On one farm, in particular, results were difficult to assess because the condition there does not always follow a typical course and instead of the affected ewes becoming worse they frequently vary from day to day, being very droopy one day, better the next, bad again the next day and so on until death or spontaneous recovery takes place.

However, considering the general picture, I should say that glycerol is definitely superior to any other treatment employed so far.

I should like to know whether any members have experience of the feeding of dried ground whale meat in this disease, because a farmer of high repute and integrity in discussing the subject stated that the position in his flock in which serious losses always occurred had improved greatly when he began feeding this product to the ewes.

He considered that not only had he prevented future outbreaks but also cut short the outbreak existing at the time.

This was several years ago when most of us were of the opinion that the condition was due to excess of carbo-hydrate and not to shortage of protein.—Yours faithfully, H. Forsyth, Lechlade, Glos. *November 14th*, 1952.

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TECHNICAL AND PRACTICAL APPLICATION OF ELECTRO-THERAPY IN EQUINES

Sir,—In an age when the horse is no longer the chief patient in most practices, it is refreshing to read the paper that Mr. Gold presented at the recent Congress and the excellence of the subsequent discussion.

I have never used electro-therapy on horses, and I have been deterred from it, because my experience of cases on which it has been used is the same as that commented on by Mr. Male and others. This does not infer that it is of no account, and it would appear that a careful selection of the suitability of the case for such treatment—as in all methods of treatment—is the criterion for success. I hope that Mr. Gold will inform us when his trials are complete, of the paste he finds the best. Armoricaine powder mixed into a paste with cold water is of value in some cases of enlarged joints capped hocks, wind galls, sprains of muscle or tendon. If a hot application is indicated, then I use kaolin or antiphilogistine. When topical treatment fails to cure a strained tendon, I turn to the cautery; not that I have ever liked it, but because I have not yet encountered anything better.

With a bony enlargement such as splint, or spavin, I have tried all so-called absorbing blisters, but in the end it usually has meant what I should have started with—pin-fire and blister.

A few years ago a member, now deceased and who had a wide reputation, advocated the administration of an electric shock to mares directly after "covering," stating that he considered that this ensured conception, "by hastening the release of the all important egg." In all probability, our ancestors, with the same view in mind, adopted a far easier and cheaper method, by tying a bunch of nettles under the tail coupled with a short sharp slap on the rump! In mentioning this, I wish to emphasise that I am *not* disparaging the treatment under discussion.—Yours faithfully, J. F. D. Tutt, "Rothiemurchus," Winchester. *November 14th*, 1952.

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ARTIFICIAL INSEMINATION IN THE BITCH

Sir,—A fortnight ago I sent you a brief and quite unimportant clinical note concerning the successful artificial insemination of a miniature sheep dog, in the belief that it might be a guide and help to other practitioners faced with a problem similar to that which confronted me.

I was the fourth veterinary surgeon consulted by the despairing owner in this particular case, and appear to have been the only one to suggest an attempt at A.I.

I therefore used the remark that "A.I. for bitches was probably only comparatively rarely attempted."

In the current issue of *The Veterinary Record* I see a letter from Mr. McGuinness to the effect that, although he had not done it himself, he had heard of others who did; he chided me for apparently believing it was a seven days wonder, and expressed surprise that I was not more *au fait* with modern treatment.

Possibly Mr. McGuinness did not intend to be rude and offensive, but his letter appeared remarkably like it. Perhaps I should apologise that after over 30 years in practice I am still not up to the McGuinness standard. Well, possibly, many of us have still a lot to learn! When Mr. McGuinness has made his own first attempt at A.I. in bitches, I shall not personally feel prompted to be offensive to him for recording the fact.

Having served on the Editorial Committee of *The Veterinary Record* for much more than a quarter of a century, I know how starved that publication has been for clinical material, and I know no surer way of deterring would-be writers from recording their clinical experiences than to have themselves and their work destructively criticised.

Are we to believe that no "clinical" can be acceptable to your readers unless it records some entirely *new* experience? Is it not a fact that the value of any method of treatment is best assessed by the consideration of numerous reports? I believe that to be the case, and I would appeal to all potential contributors to *The Veterinary Record* not to be deterred, shy, or frightened by the criticism (sometimes irresponsible, sometimes malevolent) which may follow. Personally, I do not care two hoots about it, as if I can be shown to be in error, I would realise it would be in my own interest to know it, and would appreciate constructive criticism. The other kind is hardly worthy of notice.—Yours faithfully, H. Kirk, "Croyland," 658, Finchley Road, Golders Green, N.W.11. *November 11th*, 1952.

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THAT SLANG TERM "VET."

Sir,—Lately I have read quite a few copies of your journal and have come across your weekly articles on "That Slang Term 'Vet.'"

I wish I knew what all the fuss and bother is about the very inoffensive word "Vet." It seems to me all VETS who decry the use of the word simply have bees in their bonnets, chips on their shoulders or too little to do.

To my mind it means an abbreviation of the word veterinary surgeon or veterinarian (lets face it), just as "Doc" is an abbreviation of doctor and no slur is surely intended by the great majority of people who use it. Why all frantic attempts to do away with the word? Is it impossible to regard it as a friendly term just as "Doc" is? I know you also are doctors but so as not to confuse the issue—Long Live the word "Vet."—Yours faithfully, Lyn Balfour (Miss). Avenida Juarez, 101, Coyoacan, Z.21, Mexico, D.F., Mexico. *October 31st*, 1952.

[*This correspondence is now closed.—Editor.*]

DISEASES OF ANIMALS ACT, 1950—GREAT BRITAIN

Summary of Returns of Confirmed Outbreaks of Scheduled (Notifiable) Diseases

Period	Anthrax	Foot-and-mouth	Fowl Pest	Parasitic Mange	Sheep Scab	Swine Fever
1st to 15th October, 1952	81	3	9	—	—	31
Corresponding period in—						
1951	13	—	9	—	—	112
1950	8	—	1	—	—	34
1949	11	—	47	—	1	—
1st Jan. to 15th October, 1952	738	494	180	—	1	723
Corresponding period in—						
1951	332	28	610	—	5	1,025
1950	285	20	103	—	21	210
1949	177	13	455	—	34	5

Tuberculosis Order of 1938

The number of bovine animals slaughtered under the Tuberculosis Order of 1938 during the period July 1st to September 30th, 1953, was 689.